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No. 5—Stuttgart Disease and Nephritis*

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For some time there has been a division of opinion as to whether Stuttgart disease and nephritis in the dog are one and the same disease or whether they are separate entities.

Stuttgart disease has been known by the following names: Canine typhus; haemorrhagic gastro-enteritis; dog plague; black tongue; la fièvre typhoïde.

There have been many suggested causes of this disease: anaphylaxis, intoxication, avitaminosis and infection with various organisms such as *Bacterium coli*; paratyphoid; spirochaetes.

A move in the aetiology was made when it was realised that the kidneys played an important part in the disease, and that a condition of uraemia was frequently met with.

The very name "Stuttgart Disease" seems to have been a stumbling block in the past, because it appears to be applied to several conditions the aetiology of which may be quite different. Another difficulty is that these conditions have nephritis as their chief complication, and one has to decide whether this nephritis is of primary or of secondary origin. Again, the causes which are put forward vary with the countries in which the disease is said to exist. Thus, on the Continent, it is apparently recognised that spirochaetes are the cause, while in America a disease known as black tongue[36] is said to exhibit symptoms similar to those of Stuttgart disease. The suggested cause of this is avitaminosis B_2. In this country opinion is divided—many suggesting that nephritis from any cause is Stuttgart disease, while others claim to separate the two. I include myself among those veterinarians who believe that what is known in this country as Stuttgart disease is, in actual fact, nephritis. I admit, however, that there may be an exception in what are described as "acute cases of Stuttgart disease," namely, acute haemorrhagic gastro-enteritis. Some of these cases are, without doubt, leptospiral in origin; others certainly are cases of acute uraemia with terminal gastritis; while a third group may be classified as being a condition of anaphylaxis or as of unknown origin and without involvement of the kidneys.

In 1898[4] there appeared in Germany and other countries a disease which assumed the proportions of a plague. This was first described by Klett, who observed it in and around Stuttgart.

It seems reasonable to believe, in view of recent work carried out on the Continent, that this might have been the disease which is now known to be due to leptospiral infection, and to be closely related to Weil's disease in man. Stuttgart disease[36] is said to have been observed in America for the first time in 1887, and as far back as 1850 Hoffer described a disease of dogs under the name of typhus.

Nephritis

The aetiology of nephritis, apart from possible avitaminosis B_2 and leptospiral infection, presents many problems. The chronic contracted kidney[3] which is so frequently met with at autopsy is so common a condition that one feels that there is still much work to be done both on preventive and curative lines. It is certain that variations in climatic conditions frequently play an important part in the onset of symptoms. One often finds a history of exposure to wet or cold immediately preceding an attack and many dogs develop the disease after a bath. Nephritis may be a result of injury, a legacy from distemper or other catarrhal fevers, or it may be a complication of them. Various organisms play their part in setting up the disease—amongst these may be found *Bacillus coli*, streptococci, and tubercle bacilli. Infection may be of the ascending or descending type[5]. Preputial catarrh, urethritis, cystitis may all be a cause of the ascending type, while prostatitis is considered by many to be a very frequent cause,

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the infection travelling up the ureters and setting up pyelonephritis. It is certain that an enlarged prostate gland and nephritis frequently go together. Ascending tuberculous nephritis secondary to primary lesions in the testicle or prostate is by no means uncommon[5]. Passage of the catheter may be a cause of ascending nephritis—the gum elastic type, mostly used in dogs, being difficult to sterilise. Descending nephritis takes place *via* the blood or lymph stream and may frequently be secondary to an infective pneumonia or endocarditis. Inhalation of certain poison gases can be included as a possible cause—nephritis being a common complication in humans affected with some poison gases. Absorption of toxins from the alimentary canal and certain chemical poisons may be responsible in some cases. One further cause may be mentioned, namely, arterio-sclerosis, either primary contracted type or secondary contracted type. Arterio-sclerosis is rare, but J. R. M. Innes[8] in 1930 recorded a case of each type. It is difficult to trace the origin of a lesion in the kidney, the glomeruli, tubules and interstitial tissue being so closely related that change in one must invariably affect the other.

Worm infestation must not be forgotten as a possible cause, particularly in dogs which have returned to this country from the East. A case was recorded by Henry Gray[1] in which *Eustrongylus gigas* or *Dioctophyme renale* was found in the kidney of a dog returned from India two years previously. This dog showed symptoms of nephritis. Referring to the presence of this worm, E. L. Taylor[2] states that it is found most commonly in the mink—usually occurring in the kidney but sometimes in the abdominal cavity. The length of time that elapsed before active symptoms were manifested in this case is interesting and it has been shown that some worms will remain *in situ* for several years. Renal calculi may also be responsible for setting up pyelonephritis—or even an acute uraemia, by occlusion of the ureter. Chronic nephritis is frequently insidious.

Nephritis constitutes a very large proportion of the cases seen in canine practice in adult dogs—and its diagnosis is very important, not only from a curative point of view but from a preventive one also. One sees many cases of chronic nephritis which, had they been brought to the veterinarian soon enough, might have been avoided or at least alleviated. There are many cases of chronic nephritis that can be kept going for years by careful attention to diet, and these cases are numerous amongst older dogs. One wonders whether some of these apparently chronic nephritics may in fact be cases of nephrosis[38]—*i.e.*, cases where there has been an acute or sub-acute nephritis which have made a reasonable recovery without too much injury to renal tissue, but where there is always a leak of albumin. Several of this type have been seen, in which the specific gravity has gone up from a very low to a normal one, yet there is still a considerable amount of albumin present in the urine and the patient shows no other abnormality.

Symptoms.

One of the chief causes of sickness in the dog is disease of the kidneys. Frequently one sees a dog—the majority of such subjects are adult—whose chief symptoms are frequent vomition, loss of appetite, and acute depression. This would suggest a possibility of acute nephritis, while a history of gradual loss of bodily condition, increased thirst and occasional bouts of sickness, in dogs past middle age, should lead one to suspect chronic nephritis. Many of these are termed "gastric" and have in the past been treated as such, but examination of a specimen of urine will enable a correct diagnosis to be made in both cases. The importance of urine examination cannot be emphasised too strongly, but it should be used as an aid to diagnosis only and the findings should be judged in conjunction with the clinical aspect. Nephritis may be divided into acute, sub-acute and chronic types.

The *acute type* may be seen in dogs of both sexes; its onset is usually sudden, beginning with dullness, a disinclination to move about, inappetence, and increased thirst. Usually there is a rise in temperature, which may reach 106° F., in very acute cases. This falls in a few days until it becomes sub-normal, which may indicate the onset of uraemia. An early symptom, which may not always be present, is pharyngitis—the back of the throat and mouth being a dirty colour—accompanied by a slight cough. After two or three days profuse and frequent vomition occurs. Pain in the lumbar region is marked, particularly on manipulation, the dog often resenting even the slightest pressure. A "tucked-up" appearance and arched back, with a disinclination to walk, particularly upstairs, is frequently seen. There is often constipation at first but after the earlier symptoms diarrhoea may supervene. Haematuria is sometimes present though by no means constant. Hepatitis may be a further complication[3], the mucous membranes of the eyes and mouth becoming icteric and bile salts and pigment being found in the urine. Dilatation of the sclerotic veins and injection of the conjunctivae is very marked. The mucous membrane of the mouth becomes red, particularly round the edges of the gums. Ulceration of the gums is common, especially round the canine teeth and inside the lips. A distinctive urinous odour is emitted from the mouth, due to retention of urea.

Uraemia is a common sequel and is frequently met with at this stage. It is ushered in by convulsions, staggering gait, and inco-ordination of movement; coma sets in, and finally death occurs. Recovery will take place in many cases, but the acute type may develop into the sub-acute or chronic.

Sub-acute nephritis is less severe than the acute and symptoms are not so marked. It may be a "flare up" of the chronic type. Occasional vomition and thirst are seen. Loss of bodily condition is not so great as in the acute form. Temperature is raised only one or two degrees, and pain in the loins is not so marked. The tongue has a dirty brown appearance. Ulceration of the mouth is not so frequent but the mucous membrane is inflamed at first and later becomes pale in colour. The stale urinous odour is not so noticeable. Skin lesions in the form of dry scurfy patches may be present[6]. Sub-acute nephritis frequently develops into the chronic form.

Chronic nephritis is most commonly met with in dogs past middle age and more especially in the male, though it is seen in the bitch. Its onset is slow and it may be a number of weeks before any very marked symptoms are seen; in fact, many cases are so indefinite that it is impossible to make a diagnosis without the aid of urine analysis. Early cases show only an increased thirst with occasional sickness—the vomition often occurring in the mornings only, and the vomit rarely containing food. It consists of white froth and usually follows the drinking of water. As the disease progresses the dog has such a craving for water that he fills the stomach persistently until there is a "pot-bellied" appearance. Appetite becomes variable and there is a slow progressive loss of bodily condition. The temporal and masseter muscles frequently become atrophied. The visible mucous membranes become pallid, and there is a distinct, almost characteristic, odour from the mouth; ulceration of the mouth and inside the lips is common. The gums are sore and bleed when touched.

Owing to the increased work placed on the heart, hypertrophy is common and dilatation of the left ventricle is produced, with not infrequently a systolic murmur due to mitral regurgitation. This is evidenced in the pulse[6] which is hard and bounding, and quite inconsistent with what one would expect to find in a dog whose condition would suggest a weak pulse. Oedema is not common but it does occur in a few cases where the affection of the heart is very marked. Glaucoma, according to McCunn,[14] is frequently seen in conjunction with kidney disease and any such case will merit further investigation. Change of temperament is in some cases one of the first symptoms to be noticed; dogs which were formerly placid becoming irritable, bad tempered and nervy. Eczema, either in the form of moist patches, or as a dry, scurfy condition—both of which are extremely resistant to treatment—is a frequent complication. History of stiffness and an inability to jump on chairs, or the suggestion that a dog suffers from rheumatism, should make one immediately suspicious of possible kidney disease, particularly if the dog be past middle age.

Fits, either in the nature of epileptic attacks or hysterical barking, and rushing about, are often one of the first symptoms observed; indeed, they may develop twelve months before other symptoms show themselves. Here again urine analysis will prove valuable in distinguishing these fits from others, such as those seen in true hysteria. Chronic cases ultimately terminate in death from uraemia. As in acute cases, there is a more marked desire for water, a subnormal temperature, complete loss of appetite profuse vomiting, inco-ordination, paralysis, coma and death.

Diagnosis.

This may be confirmed by examinations of specimens of urine, though the testing of one specimen only is apt to be very misleading.

In nephritis of any type there are marked variations in the composition of urine, and albumin is almost always present in some degree. In acute nephritis the specific gravity is usually raised and albumin may be present in large quantities. The reaction of urine in both acute and chronic cases is usually acid, but it may occasionally be neutral.

In chronic cases, the quantity of albumin gradually decreases and at the same time the amount of urea is diminished in the urine and is raised in the blood. This is due to damage to renal tissue and shows that the kidneys are unable to excrete urea. Urine of chronic nephritis gives a persistently low specific gravity which may drop to 1003.

The passage of urine at any time with a urea content of less than 1·5 per cent. is uncommon, while if it is less than 0·75 per cent. the outlook is serious[39].

The urea concentration test is impossible in most cases as it requires the collection of urine at regular intervals, and passage of the catheter is not desirable.

Space will not permit the detailing of urine tests, most of which, with the exception of bacteriological examination which may involve cultures, can and should be carried out by the practitioner himself. Most of the tests are easily performed and have been dealt with very ably by Professor Boddie in a previous paper[6, 9].

The testing of samples of blood will also be of considerable value, though the withdrawal of

from 5 c.c. to 10 c.c. is not always easy to carry out in the dog, but a test of the urea content of the blood will be of material help—the amount of urea varying from 50 milligrammes per cent. in the normal animal to over 400 milligrammes per cent. in acute cases[7].

The use of the ophthalmoscope will be of service in cases in which the eyes are involved[14].

A further method to establish the extent of damage to renal tissue and the rate of excretion is the use of X-ray. Pyelography, though expensive, may prove to be of great value in this direction. It consists of the intravenous injection of uroselectan "B," and the subsequent taking of pictures at intervals after the injection. The rapidity with which the uroselectan is excreted by the kidneys is an indication of the amount of damage to renal tissues. This method is employed with success in human diagnosis. The number of cases in which we (King, Uvarov and Withers) have practised this method has not yet been sufficient to justify the expression of opinion as to its effectiveness in the dog.

Stuttgart Disease

The definition given of this disease in this country in Wooldridge's Encyclopaedia[11] is: "A disease of dogs characterised by haemorrhagic enteritis, with a great tendency to necrosis of the mucous membrane of the mouth, tongue and alimentary tract."

Acute, sub-acute and chronic types are recognised.

The acute form is said to occur in dogs that have a previous history of gastric catarrh, and in old dogs who suffer from nephritis. It occurs most frequently in dogs past middle age and may appear as an epizoötic or enzoötic disease.

Wooldridge states that the feeding of healthy dogs with discharges from affected animals or portions of the stomach or intestines has frequently failed to produce the disease, though there are said to be cases on record where canine typhus has been set up. Transmission by lice or fleas has also been suspected by Wooldridge[11]—organisms, corresponding to those found in lesions of the alimentary tract, being demonstrated in such insects.

In the *acute form* onset is sudden and may prove fatal in twenty-four hours. There is refusal of food, vomiting and great depression. The vomit is at first greenish in colour, but later is blood-stained and has a very offensive odour. Temperature is at first high, but later drops below normal. There is an increased thirst and the patient vomits almost immediately after taking either solid or liquid. Loss of bodily condition is rapid and very marked. Anaemia and dehydration soon follow. The patient becomes weak and has a "tucked-up" appearance. Often there is an arched back. In the cases that live for several days there is extensive ulceration of the mouth, which first appears inside the upper lips and on the gums and sometimes on the tip of the tongue. The mouth is cold and clammy, and a catarrhal coating is deposited on the teeth, dark brown in colour, becoming blood-stained and extremely offensive; there is frequently dribbling of cold saliva. The patient becomes prostrate, and coma supervenes a few hours before death.

The sub-acute form usually begins with congestion of the pharynx and is accompanied by a slight cough. There is occasional vomiting and the appetite may be retained for a few days. Thirst is noticeable and vomition becomes more frequent. The dog exhibits signs of pain in the abdomen and may cry out if picked up. The eyes become sunken. Constipation, in the early stages, is followed by diarrhoea, which is often blood-stained and offensive. Ulceration of the mouth and dribbling of saliva are marked. The tip of the tongue becomes congested at first, but later turns brown and frequently becomes necrosed at the tip. The pulse becomes fast and weak. Temperature is raised only two or three degrees. Loss of condition becomes pronounced and the patient may finally die in a coma. Tenesmus is frequently seen, due to irritation of the lower part of the bowel.

The chronic form is met with occasionally. The symptoms consist of persistent vomiting of a yellowish viscid substance, irregular bowels, great thirst and progressive emaciation. The mucous membranes are anaemic and the temperature is normal or sub-normal. Occasionally mouth lesions, similar to those seen in the acute form, develop.

These symptoms as described by Wooldridge appear to be almost identical with those of nephritis.

Leptospiral Disease—Spirochaetosis

For some years investigators on the Continent have associated Stuttgart disease with leptospiral infection, and have noted the presence of spirochaetes (*Leptospira ictero-haemorrhagiae*) in Stuttgart disease, which is associated with an acute nephritis in addition to the characteristic haemorrhagic gastro-enteritis[20]. Buccal and lingual ulceration is not seen in every case of leptospirosis[28]. Lukes[18] in 1922, demonstrated the presence of leptospira and at the same time he informed the Brunner Biological Society that he had succeeded in proving systematically the existence of spirochaetes in Stuttgart disease. Since that time much work has been done by Wirth, Klarenbeek, Voet, Schüffner, Kotter, Schultz and others. In 1925, Okell and Dalling [10] showed that leptospiral infection was a cause of infectious jaundice in the dog. The importance of these discoveries is far

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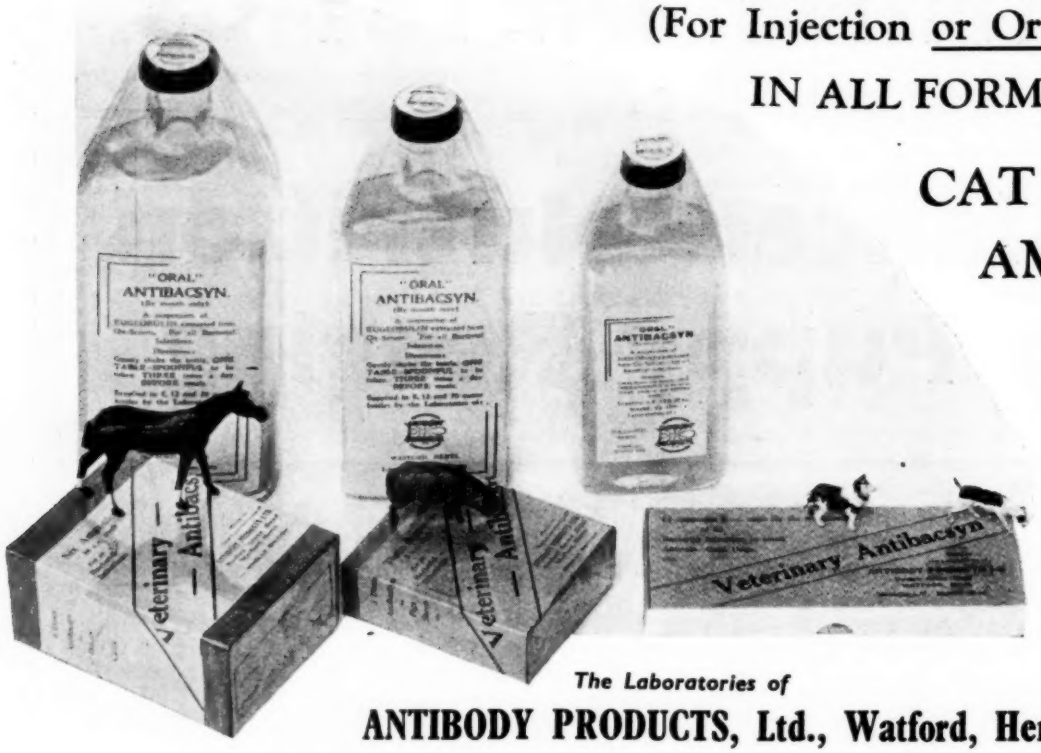

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reaching. There seems little doubt that leptospiral jaundice and Stuttgart disease are closely related, many cases of Stuttgart disease being actually leptospiral jaundice, the icterus of which is absent. There is another aspect which is important to both veterinarians and doctors—that is the close relationship of leptospiral jaundice in dogs to Weil's disease in man. The organism is carried by the brown rat and infection takes place by contamination of water or food material by urine from an infected rat—leptospira frequently being found in the liver and kidneys. Cases of humans being infected from dogs must be considered possible[22]—a serious position, if true, for the veterinary surgeon and one demanding early and correct diagnosis. How far leptospiral infection is responsible for kidney disease in the dog it is difficult to estimate, but many dogs in Germany, Austria and the Netherlands are said to show signs of acute, sub-acute or chronic nephritis together with the presence of spirochaetes either in the urine or blood. Spirochaetes have also been demonstrated in sections of the kidneys[16]. They may persist in the urine for some considerable time after recovery has taken place, probably at least a month, so that these cases are a very definite source of spread. Stuttgart disease and leptospiral jaundice appear to have a seasonal incidence which in the two diseases is similar. Old records from the Vienna Veterinary College (based on *post-mortem* returns) show that the disease is more prevalent during the summer months. The interconnection between the two diseases is further demonstrated by the clinical course and by the blood picture. There is a rise in the red blood corpuscle count and haemaglobin content, a leucocytosis, mostly affecting the neutrophiles, polychromasia and an increase in blood platelets.

Okell, Dalling and Pugh describe two types of leptospiral jaundice, hyperacute and icteric, due to *Leptospira ictero-haemorrhagiae*, but more recent investigations abroad have established the existence of another leptospira—*L. canicola*. Again, *Leptospira canicola* has been shown to be the cause in 90 per cent. of the cases in which the nephritic symptoms have predominated[15]. This organism, unlike *L. ictero-haemorrhagiae*, the organism responsible for leptospiral jaundice and Weil's disease, does not cause jaundice[13] and according to reports is not found in the brown rat.

Some interesting experiments have been carried out in conjunction with the two diseases. Wirth[25] found spirochaetes in the kidneys of 14 cases of leptospiral jaundice. Kidney suspensions from two cases transmitted the disease to puppies after subcutaneous inoculation. Similar suspensions from cases of Stuttgart disease were inoculated into seven puppies—four of which developed leptospiral jaundice. Spirochaetes were seen in the kidneys of these puppies after death. The infection was passaged several times in other puppies. Puppies which were inoculated developed the disease in 10 to 18 days and died at 19 to 23 days. He also produced the disease in puppies by the subcutaneous inoculation or ingestion of urine from wild rats taken from a canal in Vienna. Leptospiral infection has been met with fairly frequently in the Netherlands[21] and though both *L. ictero-haemorrhagiae* and *L. canicola* were found, *L. canicola* was much the more common one. Identification was confirmed either by isolation of the organism or more frequently by serological tests.

Diagnosis of leptospiral infection is not always easy; the organism may be found in the urine or blood, its presence in either depending on the stage which the disease has reached. In the later stages, *i.e.*, when the disease is approaching the chronic stage, many of the symptoms will have disappeared and one will have to rely almost entirely on examination of the urine for the presence of the organism. Leptospira may be found in the blood at the third to the fifth day and in the urine from the seventh to the tenth day[12]. Their numbers may vary in the urine and there may be few or many. In cases where the urine is acid there may be very few—the acid hindering or preventing the secretion of live leptospira[12]. This is, perhaps, an important factor in the treatment. Diagnosis[22] depends on the demonstration of the organism by direct or indirect means:—

1. The examination of urine or blood by dark field illumination.
2. The measurement of the lytic power of the blood for leptospira.
3. Agglutination tests (in two cases reported the blood showed agglutinins for a number of strains).
4. Culture of the organism in special media.
5. Guinea pig inoculation—leptospira being subsequently found in the blood of the inoculated animal.

Guinea-pig inoculation is not always successful—a difficulty being found in effecting the first passage in *L. canicola* infection.

Symptoms of Leptospiral Disease.

There is a rapid febrile course, extreme prostration and depression, complete loss of appetite and great thirst. Often there is passage of blood-stained mucus-coated faeces, vomition of bad-smelling material, haemorrhages from the lips, gums and nose. Dyspnoea frequently occurs with a short cough. Death may take place in a few hours, or the dog may live for several days. Icteric symptoms are only occasionally met with. Urine is always dark coloured and passed in small amounts. Albuminuria is invariably present. Specific gravity falls to

1005[18]; the reaction is always acid; renal cells, some leucocytes and sometimes casts are present. The quantity of blood urea is much increased. *Post-mortem* examination shows very marked lesions—a haemorrhagic gastro-enteritis—the contents of the intestine and sometimes of the stomach being bloody and chocolate coloured. There are ulcers in the buccal mucous membrane, and alterations in the renal tissue—an acute interstitial nephritis being present. Although nephritis is almost always a constant complication it is not of primary importance in *post-mortem* diagnosis[24]. Icterus may be absent[28]. The cardiac endothelium, particularly on the left side, degenerates, and this is followed by desquamation and a tendency to thrombus formation. The myocardium shows diffuse haemorrhages and infiltration. Chronic interstitial nephritis is a common sequel in those cases that do not prove fatal in the acute stage. Chronic cases show similar lesions which are older and may have undergone calcification. Some cases may show lesions of both acute and chronic types.

Black Tongue

This disease is seen in America and is said to be allied to Stuttgart disease.

Recent experiments have shown this to be due to a deficiency in vitamin B_2[29], and to have produced a condition resembling pellagra in humans[35].

In 1907, Chittenden found that dogs deprived of meat and milk fell ill with salivation, stomatitis and diarrhoea, which is so characteristic of the condition known as black tongue, and met with most commonly in the Southern States of North America. Certainly the disease picture in naturally occurring cases is very similar to that of nephritis. Cases are described as being of two types—those with and those without nervous symptoms together with skin lesions. Nervous symptoms which manifest themselves by convulsions of the clonic type invariably prove fatal.

Age, sex and breed appear to have no noticeable influence on susceptibility[36], though in a report from one clinic it is interesting to note that hounds, pointers, setters and collies from suburban and rural districts constituted 85 per cent. of the patients treated, and that their average age was $3\frac{1}{2}$ years.

All experimental attempts at artificial transmission have failed and all efforts to make cultures from heart blood have been negative[31].

Blood and urine analysis of specimens from cases revealed an increase in non-protein nitrogen, urea nitrogen, uric acid, and a fall in the chloride content of the blood. Urine was high in nitrogen and low in chlorides and contained albumin and casts.

H. C. Rea[36] records the treatment of 255 cases which included yeast, sodium chloride and glucose, lime water and calcium gluconate, carotene extracts and glycerine, and liver extracts. Liver extracts with sodium chloride appeared to give the best results and feeding of raw beef and tinned salmon was said to be beneficial.

There are several points that seem to stand out in this disease. First, the similarity of symptoms to those of nephritis, together with the blood and urine reports. Secondly, the fact that at one clinic the majority of cases were those of sporting dogs, *i.e.*, dogs most likely to be subjected to exposure to weather conditions, such as wet and cold (a frequent contributory cause of nephritis). Thirdly, the treatment recommended was similar to that which is adopted for nephritis.

One is inclined to ask "Is this a true avitaminosis with secondary nephritis, or is it nephritis with secondary avitaminosis?" A combination of the two is possible and it has been suggested that a deficiency in vitamin B_2 may be purely a secondary one due to faulty absorption—often a result of protracted gastro-intestinal disturbance, such as would be seen in nephritis[32].

The experimentally produced black tongue seems to differ from the naturally occurring cases, and much more closely resembles human pellagra.

Work on this subject has been done by Chittenden, Goldberger, Wheeler, Blackstock and others. Rhoads[34] and Miller[37] in 1932 succeeded in producing experimentally a condition of chronic black tongue with anaemia, by means of a diet almost free from vitamin B_2. Control animals which were given 4 grammes daily of liver extract together with the same diet remained in perfect health throughout, and examination of their blood failed to show any significant variation.

Interesting changes in the mouth were noted—particularly of the tongue. There was anorexia and diarrhoea and often blood was passed. Loss of weight was striking—being about 20 per cent. of the original.

The general results obtained were characteristic of the macrocytic anaemia of sprue and pernicious anaemia in man[34]. The prophylactic and therapeutic properties of foods rich in vitamin B_2 were very marked. Yeast, milk, and liver extracts are rich in vitamin B_2[33].

Treatment

Warmth and attention to diet are essential. Foods rich in protein should be withheld. Barley water should be given instead of water and limited in quantity. Milk, cream, glucose, honey, Benger's Food, etc., are all good, while injections of glucose saline and calcium gluconate

will be found particularly helpful in cases of dehydration and collapse.

Bread and butter, and milk puddings may be given after the acute stages when vomition has ceased, and fish, poultry and boiled mutton may be given in small amounts as the patient is convalescing. Alkalis such as sodium bicarbonate will prove useful and combinations of bismuth will be of value in checking vomition. Salicylates are useful in relieving pain. Attention should be paid to the heart by the administration of digitalis, nux vomica, caffein and camphor in oil and ether. Nervous symptoms may be controlled by sedatives and respiratory changes in uraemia may be helped by the injection of calcium gluconate. Rectal injections of normal saline solution may alleviate straining when haemorrhagic enteritis is present.

Mandelic acid[30] has been used as a urinary antiseptic in the treatment of nephritis, but it is suggested that it should not be employed when the renal function is impaired. Intravenous injection of formalin has also been used[3].

In leptospiral infections a polyvalent anti-spirochaete serum has been used with success. Kantorowicz[23] records seven cases in which serum was given in Stuttgart disease and appeared to be of curative value. Various other medicinal aids have been used in leptospiral infections with variable results. Amongst these are pallicid, introcid given intravenously and salvarsan[17]. Rudolf and Mendal[27] report 100 per cent. success with magnesium perhydrol administered frequently and in small doses.

CONCLUSIONS

Nephritis, from various causes, is a common condition in this country.

Haemorrhagic gastro-enteritis is frequently leptospiral in origin.

Black tongue in America appears to be associated with kidney disease, though in the experimentally produced condition the kidneys do not appear to be involved.

"Stuttgart disease" is a name that has been applied to all three conditions.

Nephritis is undoubtedly a common complication of the condition known as Stuttgart disease in this country, and is frequently seen in leptospirosis on the Continent.

Leptospirosis (producing acute haemorrhagic gastro-enteritis) is known as Stuttgart disease abroad.

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The first paper to be presented to the section of Veterinary Medicine and Surgery was on the subject of "Stuttgart Disease and Nephritis" which was considered by a good attendance in the Grand Hall at the Town Hall, Bournemouth, at 11 a.m. on Wednesday, July 28th, 1937.

The President of the section, Mr. J. F. D. Tutt, of Southampton, introduced the essayist, Mr. N. S. King, of Leatherhead. Mr. King observed that he did not think that he had very much to say by way of introduction to the discussion of his paper, except to express his appreciation of being invited to prepare that paper for the "National."

The subject was an extensive one and one which was very much before the canine practitioners, but he was afraid that he had had to omit some branches of it, or at least curtail very considerably, the number of words he was allowed being so limited.

One of the points that he had omitted was in reference to the treatment of nephritis by vaccine. He did not desire to enlarge on that at all, however, because he was sure that Dr. Hare would be speaking on it, and as the latter had had very considerable experience of that treatment, he did not propose to say anything further concerning it or in regard to the paper itself.

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Discussion

Professor G. H. Wooldridge, F.R.C.V.S., M.R.I.A. (Vice-Principal, Royal Veterinary College, London), who opened the discussion, said:—

It has been remarked with a certain amount of cynicism and a considerable amount of truth that it has become the fashion to commence a discussion on a thesis by complimenting the writer on his efforts, whether deserving or not. I have no hesitation on this occasion in congratulating Mr. Nevill King on at least two things—namely, his great courage in acceding to the invitation of the Provisional Committee to submit a paper on the subject of "Stuttgart Disease and Nephritis" knowing the great diversity of opinion in respect of these two conditions, and secondly, on the thorough manner in which he has undertaken the task. He has certainly presented an excellent case for discussion.

I said "these two conditions" and here at once is the kernel of the discussion. Are they "two conditions" or is such an idea only a delusion? Mr. King appears to make his position quite clear early in his paper, for he says: "I include myself among those veterinarians who believe that what is known in this country as Stuttgart disease is, in actual fact, nephritis." That appears to be very definite, but it is immediately followed by a statement that completely obscures the apparent clarity of that remark, for the writer says: "I admit that there may be an exception in what are described as 'acute cases of Stuttgart disease,' namely, acute haemorrhagic gastro-enteritis." But I submit that he cannot have it both ways. Again at a later stage he says that many cases of Stuttgart disease are actually leptospiral jaundice. Surely he does not suggest that leptospiral jaundice is also nephritis, though nephritis may complicate the picture.

It must be admitted at once that many cases of nephritis in dogs with ulceration of the buccal mucosa and with vomiting and severe terminal congestion of the gastric and intestinal mucous membranes have been wrongly diagnosed before death as Stuttgart disease, and apparently, though mistakenly, confirmed on *post-mortem* by the condition of the stomach and intestines. Dr. Hare has emphasised the existence of this acute terminal congestion in nephritis and its proper interpretation, but I have not heard him deny that Stuttgart disease may occur as a separate entity from nephritis.

It is a curious fact that a number of diseases appear to change somewhat in their manifestations as time passes on. There may be a wave of great severity followed by a period of less severity and almost a spontaneous disappearance of the condition. I suggest that that almost applies to Stuttgart disease. I rarely see a case of Stuttgart disease now of the type we used to see, but we had a case this summer (which proved fatal) in which *post-mortem* examination of the kidneys failed to reveal any macroscopic evidence of nephritis. Of course microscopic examination is essential to determine whether a kidney is normal or not in many cases, but I submit that a fatal acute nephritis would show macroscopic changes.

New ideas suggest that the old practitioners were sadly ignorant, and in the light of present knowledge there may be much justification for that opinion, but they were not without powers of observation; indeed, I venture to suggest very strongly that their powers of observation were much better developed than in most of the younger generation, who have unfortunately developed a practice of relying too much on laboratory assistance and allergic and various biological tests to the exclusion of their faculties of observation. Do not misunderstand me—I do not in the least decry laboratory assistance. I advocate it very strongly and I myself resort to it probably more frequently than most people, but the findings must be interpreted in conjunction with the clinical symptoms as discovered by careful observation and examination of the actual patient. In fact, the clinician and pathologist must work together. They are complementary and it is team work of this kind that will give the best results.

The older generation who met with so-called Stuttgart disease in the last few years of the 19th and beginning of this century were also conversant with nephritis and it was very obvious that they were entirely different. In fact, the more acute cases were very suggestive of acute phosphorus poisoning. I have only occasionally seen this old type of Stuttgart disease in recent years, but I frequently meet with cases of nephritis with vomiting and with ulceration of the gums and buccal mucosa and extremely offensive in odour. There has been a tendency on the part of those who have not seen the real thing to suspect and to diagnose this wrongly as Stuttgart disease. That wrong diagnosis certainly does not make Stuttgart

disease and nephritis one and the same disease, rather is it a mistake in differential diagnosis. It is a good sign, up to a point, that some of those who have made that confusion are now realising that they were really dealing with nephritis. I fear the essayist is one of those and from his report I begin to wonder if he has ever seen a true case of Stuttgart disease; if he has not, that can be no ground for asserting its non-existence as a separate disease unless he has the temerity to accuse his predecessors of lack of the powers of observation which I claim for them.

There was one characteristic feature in acute and subacute Stuttgart disease which I have never, in the whole of my experience, seen due to nephritis, and that is the sloughing of the tongue followed by recovery. I have seen the whole of the free portion of a dog's tongue slough off in Stuttgart disease and the dog on recovery learn to drink like a horse by immersing his mouth into water provided in a vessel deep enough to permit it, but unable to drink at all from shallow vessels.

I observe that in giving what is suggested as a summary of symptoms of Stuttgart disease from my contribution on the subject in the Encyclopaedia, the essayist simply states that: "The tip of the tongue becomes congested at first, but later becomes brown and frequently becomes necrosed at the tip." That does not convey the clinical picture intended by the actual words in my account, namely: "The tip of the tongue becomes slightly congested, and then brown and this is followed by necrosis which is indicated by the tip or edge of the tongue becoming yellow. This portion may fall off and in some cases the dog may actually lose the entire free portion of the tongue." It would appear that the essayist has relied largely on his paraphrasing and summary of my description of Stuttgart Disease in the Encyclopaedia to justify his statement that: "These symptoms described by Wooldridge appear to be almost identical with those of nephritis." It would have been more correct to say, "This train of symptoms extracted by me from Wooldridge's account of Stuttgart disease is almost identical with those of nephritis," but that would have been begging the question. Then again, nephritis in adult dogs is very common, and it might well happen that a dog already affected with that condition might contract Stuttgart disease, but this would not make the two affections one and the same disease. In my view there is a severe intoxication in both affections, and it is not surprising that many symptoms are met with in common. Apart from that I cannot accept his conclusion.

By the way, I note Mr. King states that "An early symptom (of nephritis) which may not always be present is pharyngitis." I must confess that this conflicts with my experience, and I doubt its accuracy. If it is a fact, perhaps Mr. King will explain how it comes about, and I would ask why pharyngitis and necrosis of the tongue should occur in nephritis in dogs and not in other species.

To me, however, the most interesting part of Mr. King's paper lies in his detailed reference to leptospiral disease and its resemblance to Stuttgart disease, and I am inclined to agree with him when he states, "There seems little doubt that leptospiral jaundice and Stuttgart disease are closely related, many cases of Stuttgart disease being actually leptospiral jaundice." The result of my observations on the epizootiology of Stuttgart disease led me to suspect that a vector of some type was responsible for its spread, and I have suspected lice and fleas. The circumstances did not exclude the possibility of rats—rather the contrary—and as rats are known to be carriers of leptospira it opens up fresh possibilities in respect of Stuttgart disease. Indeed, I would suggest the use of leptospiral serum in the treatment of early cases suspected of Stuttgart disease, as a precautionary measure, owing to the great possibility of their association.

I am afraid I have already taken up too much time, and have not been able to cover the whole course of the paper nor the question of treatment. The paper is packed with useful information and references, and contains much food for thought and discussion.

I must leave the remainder to other speakers, but in summing up, I would suggest that Mr. King in one respect resembles Balaam of old who set out with a certain purpose and accomplished the opposite. Mr. King set out to prove the identity of Stuttgart disease and nephritis, but in my view he has gone much further to support the identity of Stuttgart disease and leptospiral disease, though I must regard this at present as not fully proven.

Dr. T. Hare (London) said that he was just cudgelling his brains as to how to build a bridge between his old friend Professor Wooldridge and his colleague Mr. King, who, with Miss Uvarov, had done so much to translate into practice some of the notions he (Dr. Hare) taught in his R.V.C. days. He knew from experience the great value of Professor Wooldridge's clinical acuity. He felt both were right and that a bridge could be built between them. He was just thinking how to do it. He would start with the clear statement that in all cases submitted to him as Stuttgart disease, on detailed microscopic examination not only of the kidneys but of the nervous system as well as the other internal organs, he had been unable to determine a specific entity, Stuttgart disease; the cases proved to be nephritis. Of course he had not had the opportunity of meeting with those cases which Professor Wooldridge used to see at the College 20 or more years before the War. It was possible, as Professor Wooldridge had pointed out, that the disease might occur in waves or cycles and that the one-time common disease had almost disappeared from our dog population to-day.

He would ask them to consider that what Mr. King was speaking of as clinical nephritis might be produced by a variety of infectious diseases. Nephritis was not a specific entity; it was a morbid state. It might follow distemper, gastro-enteritis due to worms or coccidiosis, leptospiral jaundice and other specific infections, particularly those due to haemolytic streptococci. He and Dr. Fry hoped very soon to publish a report of their first two years' work upon canine infections due to Group G beta haemolytic streptococci. This group of streptococci was the cause of puerperal sepsis in the bitch, and contagious sore throat of either sex. The mortality rate of this Group G haemolytic streptococcus was low, but the morbidity was very high. The puerperal sepsis resulted in abortion or death of puppies within a few hours or days of full-term birth; the bitch herself was but mildly upset as a rule, occasionally she died of septicaemia or pneumonia. But the clinically recovered bitches retained the infection; they were carriers; and they became sterile. This carrier question and the sterility resulting from the infection were making it almost impossible for some pedigree breeding kennels to continue in business. He regarded this as one of the most serious infections in the canine world to-day.

He and Dr. Fry had also met with occasional cases of Group C and Group A beta haemolytic streptococcal infections in dogs; the Group A infections were of concern to the medical profession since they were the common cause of human puerperal sepsis. These streptococcal infections were of importance to the present discussion since they commonly resulted ultimately in nephritis.

There was another difficulty which had received insufficient attention—the differentiation of pyelonephritis from the true Bright's disease type of nephritis which Mr. King had been describing. In pyelonephritis the infection came up from below, not *via* the blood stream. He believed that differentiation to be of vital importance to clinicians for this reason: that in the descending or Bright's type of nephritis he was afraid the utmost they could do clinically was to study the diet and the other principles indicated by Mr. King, to make the dog's life reasonably comfortable for some months or possibly years. The dog's death certificate had already been written by that diagnosis: the scarred kidneys were eventually going to kill him. With pyelonephritis, the picture was entirely different; without qualification, he said that these were cases to be cured provided they got them in the stage before the cortex of the kidney had been involved and destroyed. These cases of pyelonephritis should be treated by employing the whole armamentarium of clinical treatment combined with autogenous vaccine therapy. The differential diagnosis must be established by microscopic and chemical examination of the urine and the examiner must have had some years of experience at his job. Clinicians could approach the cure of pyelonephritis with confidence and could give a good prognosis to their clients; though it would take them six months before they cleared up the disease. They had to continue the vaccine treatment at intervals until they had got the albumin completely absent from the urine.

He believed, therefore, that a bridge could be built between their two protagonists. None of them could deny the vast experience of Professor Wooldridge: he was looking back to a period when he (the speaker) was a child and it might be that in those earlier days there was a specific entity which to-day they did not meet with. But none of them could prophesy; it was as well to have Professor Wooldridge taking that attitude and they hoped he would always take it, because it would prepare them for the possibility that the specificity of Stuttgart disease which he held so sincerely, might crop up again, and then the profession would be prepared for it.

Mr. H. Raynor Hewetson (Southport) said that they must all have considerable sympathy with Mr. King in the very difficult task he had had to undertake, but he hoped he might also claim a little sympathy for himself as he had come to the meeting without any notes and had, therefore, not intended to speak. Since he had been called on unexpectedly to do so, he hoped they would forgive him if his observations were somewhat brief and incoherent.

Whilst congratulating Mr. King on his paper, which evidenced both wide reading and careful observation, the first thing that struck him was that Mr. King had not lessened his own difficulties by not taking a strong line in defining which condition he meant by Stuttgart disease. If three different conditions were to be described and then lumped together and referred to as Stuttgart disease, it became impossible to understand what one was expected to compare with nephritis. This loose grouping of a variety of conditions presenting some symptoms in common under one name was one of the prominent weaknesses of veterinary practice to-day. It might be that the demand of owners for a name for the patient's condition was responsible, but this expediency would be better avoided in a scientific discussion.

Taking nephritis: this was a vast subject, it was a symptom of so many conditions and he did not feel they could properly discuss it on the lines of the effect on the kidneys of various infective and toxic agents.

His own view was that when talking about nephritis they should separate the disease itself, where the effects and symptoms were the result of purely pathological or organic changes in the kidney, from those secondary renal inflammations consequent on bacterial infections or toxaemias. Unfortunately there was very little pathological data correlated with clinical symptoms to go on, and he felt that nowadays pathology was simply being swamped by bacteriology. Mr. King had put forward several causes for Stuttgart disease—or rather had described several conditions as Stuttgart—but had omitted to mention what he (Mr. Hewetson) could remember used to be considered a cause, distemper. In fact, 15 to 20 years ago many people held that Stuttgart was a "form" of distemper. More recently Pugh had described "infectious brown mouth" and in a great many ways this coincided with Stuttgart disease, except perhaps in the nervous symptoms which followed in the train of "brown mouth."

Professor Wooldridge had said that typical Stuttgart appeared much less frequently nowadays than it used to do. He (Mr. Hewetson) agreed with him and said it was quite a number of years since he had seen a typical case of what he regarded as Stuttgart, but which at one time he saw frequently, and at that time the symptoms had been unmistakable and constant. His own view was that Stuttgart disease was really an anaphylaxis to some unclassified organism which gave rise to a stomatitis, gastro-enteritis and nephritis, but with a special point of attack in the tongue and mouth where it caused necrosis and sloughing. It might be that

grouping of the organisms in the finer capillaries in these regions not only did damage by their toxic action but also by interference with the blood supply, causing local death.

In Stuttgart, as in many other diseases, notably distemper, the nephritis which was observed was a secondary condition, due either to the damage done to the kidney by the organism itself, or to the breakdown of the parenchyma from the strain of the overload on its functions.

Lieut.-Colonel H. Greenfield (Camberley) said that it was indeed a pleasure to rise and congratulate the essayist upon an attempt to elucidate the cause of nephritis, of which we knew so little, for it appeared to him that the paper was really one on nephritis considered from the recent work on Stuttgart disease and leptospiral jaundice.

The author showed a desire in his paper to associate these three conditions or classify them together, but he did not support the case by the suggestion that nephritis was a consequence of exposure to cold, with which many did not agree, while he pointed out that Stuttgart disease was a pathological condition met with in the summer.

On the other hand, the inclusion of a brown tongue and ulcerated mucous membranes of the mouth as symptoms of nephritis would link the diseases together, but the speaker, and he thought others, would not agree to these signs being descriptive of nephritis.

While many would be prepared to admit that Stuttgart and leptospiral jaundice included symptoms of nephritis, the outstanding features of Stuttgart were those of a lifelessness due to a toxaemia, specific in type, with a foul, sewer-like smell to the breath, teeth and tongue coated brown, with a fixidity of the jaws difficult to overcome until the animal sank into a sub-normal temperature and pulse, and semi-coma—the fatal ending yielding *post-mortem* symptoms of the typhus type. Few if any of these were characteristic of nephritis and while they frequently saw many cases of the three conditions occurring simultaneously, they rarely, if ever, saw the apparent outbreaks of nephritis, leptospiral jaundice or Stuttgart disease at the same time.

Mr. King had fallen back upon the literary differentiation of nephritis into acute, semi-acute and chronic. That always sounded right and nice, but in actual practice it was frequently very difficult, and the change from one type to the other seemed rare although described in literature as the reverse. He went on further to suggest that there were two forms of Stuttgart disease, one of which was nephritis and one not.

In his (Colonel Greenfield's) opinion, there seemed a much more definite differentiation, namely, that there were two forms of nephritis, one infectious in type and one insidious and not infective, and that it was the former which had a similarity or relationship to Stuttgart. One frequently saw the former in considerable numbers together occurring in dogs of any age with a rapid onset, depressed demeanour, violent symptoms and continued vomiting, often almost irrepressible. In the latter form almost the reverse was found. It occurred in isolated cases, nearly always in old dogs, with the owner suddenly surprised at a continued depreciation of weight and condition because the animal was so bright and cheerful, and still more surprised at the seriousness of the occasional vomition at night or in the morning before food, which was such a decisive symptom.

A similar contrast was found at routine urine testing. The infective type showed a large dense precipitate to nearly all reagents, generally of rather a yellowish colour, while the insidious type showed urine of a very low specific gravity and colour with a trifling precipitate of a bluish tinge—and the less there was, the worse was the prognosis of the case. He would like to ask the essayist if he concurred with this opinion as to the type colour of urine and precipitates at testing.

The speaker, brought up in the routine school of Sir Frederick Smith, doing urine tests constantly, disagreed with the author that the urine was always acid in nephritis, as he disagreed at a former discussion on Mr. King's paper presented to the Central V.M.A. He found the reaction to vary from alkaline to neutral to acid in different cases and from day to day in the same case.

As on that occasion, he (Colonel Greenfield) still maintained that the insidious chronic nephritis was due to toxaemia from alimentary disorders, chronic enteritis, etc., and that its incidence could be foretold in animals known intimately under former treatment, while that of the more acute type could not be so anticipated, and he thought they were due to an infection—possibly distemper, in many cases.

Finally, he would like to report that for the past few months he had been treating nephritis cases by a system which he could only describe as shock treatment, to obtain a non-specific protein reaction by the use of intravenous injection of formalin, the subcutaneous use of omnadin or S.U.P. or the intramuscular injection of two-and-a-half to four ounces of sterilised milk. With these, especially the last named, he had obtained a quick response, checked vomiting and got better results than ever before.

Mr. A. R. Smythe (Falmouth) said that he thought Mr. King's paper was the most interesting one he had read on canine medicine for quite a long while. It opened up a big subject, and to start at the beginning of his experience, every year, both in London and the provinces they met with epidemics in dogs characterised by what one might term a marked attack of gastritis, in the first place. Those cases went on for four or five days and the majority resolved themselves and became convalescent. About 6 or 8 per cent. developed Stuttgart and some developed nephritis. He had recorded those epidemics on several occasions in the journal of the Association. The cases of Stuttgart might be of the peracute form: they died before one really realised that one was treating Stuttgart. Alternatively, the case might develop into one of the type mentioned by Professor Wooldridge characterised by necrosis of the tongue. The nephritic form was rather interesting in that they got those cases brought to them again. They ran a temperature, the specific gravity of the urine was low, around 1010, and there might or might not be traces of albumin. These dogs did not respond to treatment readily and he thought that the kidney was the source of the trouble and that the kidney condition was secondary and a complication of the primary condition which was infective. He did not think that Mr. King would confuse true nephritis of the chronic form with Stuttgart disease, as canine practitioners knew nephritis only too well.

While talking about that subject he would point out that the presence of albumin in the urine did not necessarily imply that the dog was going to die. He had known dogs with large quantities of albumin in their urine for the last two or

three years and they remained in apparent health. As to leptospiral infection, that occurred in two forms: the true jaundice and the type where jaundice was absent. Probably the kidney condition in both these types of jaundice was due to an infective process.

He had endeavoured to show that many of the kidney abnormalities met with in canine practice were due to complications associated with various infections.

The Reply

Mr. N. S. King, replying to the points raised in the discussion, said that when he heard that Professor Wooldridge was going to open the discussion on that paper, he was reminded very much of Jonah and the whale. (Laughter.) Professor Wooldridge had "called him over the coals," perhaps, for one of his earlier remarks about the differentiation of Stuttgart disease and nephritis. One of the primary objects that he had in mind when he set out to write that paper was to try and set some sort of standard before them: the whole subject of Stuttgart disease seemed to be such a muddled one, in his opinion. He admitted that he might never have seen the true Stuttgart disease; he did not think that he could have done. At the same time, the term "Stuttgart disease" was, on the Continent, applied to a very definite infection, namely, leptospiral infection and he had had it in mind to try and separate those two or perhaps three conditions.

Professor Wooldridge had raised the question of the difference between Stuttgart disease as described by him and nephritis. Nephritis might be, or might have been, a complication of the old suggested Stuttgart disease; in fact, Professor Wooldridge said in his book "The acute form is said to occur in dogs that have a previous history of gastric catarrh and in old dogs which suffer from nephritis. It occurs most frequently in dogs towards middle age and may appear as an epizootic or enzootic disease." He thought that rather bore out his own opinion.

Concerning the question of sloughing of the tongue, he could not at the moment find that passage in his paper, but he thought that there probably was some error there. If he said "sloughing" he meant sloughing of part of the tongue and he had seen a number of acute cases of nephritis where there had been very definite necrosis of the tip but not of the whole tongue.

He must apologise to Professor Wooldridge for the paraphrasing. He (the essayist) did not base his remarks on his (Mr. King's) paraphrase but on the actual words in Professor Wooldridge's book and he (the essayist) tried to cut Professor Wooldridge's remarks down as much as possible, realising that the subject was a very extensive one.

Professor Wooldridge also mentioned a case of acute Stuttgart disease and that the pathological and microscopic examination proved negative so far as involvement of the kidney was concerned. He wondered if the Levaditi silver impregnation method of examination of the kidney was carried out, because it was possible from Professor Wooldridge's account that that might have been of leptospiral origin. Leptospira were apparently very difficult to demonstrate in some cases, and in immersing the kidney tissue in a preparation of formalin, some workers found that the longer the kidney tissue was immersed the more likelihood there was of demonstrating the parasite.

He had found pharyngitis quite common and his explanation of that was that probably the whole thing was an infection. It might be that in these cases the nephritis was secondary to infection with haemolytic streptococci or some other organism and the pharyngitis was perhaps the first symptom to be shown. One wondered how Professor Wooldridge accounted for his observation of pharyngitis in Stuttgart disease.

Dr. Hare raised the question of the disease dying out. He thought Dr. Hare had perhaps solved one of his difficulties there. He agreed that in human medicine one got changes of type in diseases and quite possibly the old type of Stuttgart disease might have died away for the time being.

The question of pyelonephritis and interstitial nephritis, of course, was an interesting one. They knew that the life of the chronic interstitial nephritic was doomed eventually, but that one could keep them going for some time. If one was dealing with pyelonephritis, however, he thought there was quite a field for treatment with vaccine which would probably help them very considerably.

Mr. Hewetson had remarked about Stuttgart disease being secondary to distemper. He had said in his paper that he considered that nephritis might be associated with a catarrhal condition and if one assumed that nephritis and Stuttgart disease were the same condition or that nephritis was a part of Stuttgart disease, then the same applied to that. Brown mouth did, from the reports, appear to have some relationship with the so-called Stuttgart disease; at the same time, there was again, apparently, frequently involvement of the kidneys. He did not mention it at all in his paper because he could not see that they had recognised it as such and there had been very little in the literature so far.

Colonel Greenfield had remarked on exposure to cold and wet. He still thought that cold and wet were very predisposing causes; he did not say that they were the direct cause, but one so frequently found cases of nephritis following on wet and cold. He thought it was a question of lowered resistance.

Regarding the seasonal incidence of Stuttgart disease, he had referred to that as leptospiral in origin. The changes in the mouth, the brown coating of the teeth and the discoloration of the tongue he admitted one did not see in the early cases of nephritis but in acute nephritis one did get very distinct inflammatory changes to begin with. When he saw the edges of the tongue quite early on become reddened and the line around the gums also becoming reddened, he immediately suspected nephritis, but one did not base one's opinion entirely on clinical examination and in such cases urine examination, of course, was a great help.

Concerning the question of alkaline and neutral reactions of urine, he had found that neutral reactions were rare and that alkaline reactions indicated cystitis. One did occasionally get an alkaline urine but an alkaline reaction in the dog might be due to the samples being old, *e.g.*, specimens having been kept for some time. He could not say that he had noticed the variation in colour of the precipitate at all.

Mr. Smythe had raised the question of the albumin in small quantities with a fairly low specific gravity and he thought, as he had said in his paper, that some of those cases might be due to a nephrosis. He had got some that were alive now; he was waiting for them to die, he was afraid, so that he could have histological examinations made: cases where one had had an acute nephritis, sometimes running on to subacute, in which one got apparent recovery. Then,

MILK POLICY

The Government's Programme

Under the heading "Milk Policy" there has been issued an official publication* in which is detailed the projected milk policy of the Government—a matter of such moment to the profession that we reproduce the text in full, as follows:—

(1) The Government have carefully considered the position of the dairying industry from the points of view both of producers and of others engaged in the industry and of consumers. They have had before them the Report of the Reorganisation Commission for Milk, other authoritative Reports bearing on the industry and also the views of representative organisations, all of which have been of the greatest assistance. They have also taken into consideration the recent outcome of the application made, on behalf of milk producers, to the Import Duties Advisory Committee, for increased duties on butter and cheese.†

(2) The Government desire to see the dairying industry of this country self-supporting and profitable and their policy for this industry, as for other branches of agriculture, is to ensure the maximum supplies for the consumer at fair prices consistent with reasonable remuneration for the producer. They believe that the only sure foundation for the prosperity of the industry is an increased consumption of liquid milk. An essential step towards this objective is to establish public confidence in the cleanliness and purity of the milk supply and thereby to quicken demand for what is one of the most valuable of human foods. It is with this object in view, and in order to bring to full achievement the efforts that dairy farmers have themselves been making to improve the quality of their product, that they propose to invite Parliament to provide additional Exchequer assistance on the lines set out below.

(3) The Government also propose to continue and extend their policy of promoting the increased consumption of milk among those sections of the population for whom it is of particular value.

(4) With the approval of Parliament, the dairy farmers of Great Britain a few years ago set up Milk Marketing Boards under the Agricultural Marketing Acts. In this way, they were able to cope with a price situation that had been steadily growing unstable and to avert a collapse which would have spread disaster through the countryside.

(5) The industry, thus enabled by Parliament to attack its internal problems, was further assisted by a temporary Act called the Milk Act, 1934, the main provisions of which expired in 1936, which had a three-fold object, (*i*) to supplement the low receipts of the Milk Marketing Boards from the sale of milk for manufacture, (*ii*) to increase the consumption of liquid milk and (*iii*) to promote a purer milk supply. The first was of an emergency nature; the second and third indicated the lines of future policy. This measure has been extended once, and by the Milk (Amendment) Bill of this Session, it is proposed to extend the main provisions for a further period until September 30th, 1938.

(6) It is still desirable to safeguard the industry against emergency conditions, but circumstances have altered to an extent that now enables the Government to lay relatively greater emphasis on measures designed to promote the increased consumption of liquid milk and the provision of a purer milk supply.

(7) Under the Milk Act, 1934, the receipts of the Milk Marketing Boards from manufacturing milk were supplemented by a system of repayable Exchequer advances based on standard prices per gallon of milk used for manufacture. The Government now propose to safeguard the industry against the effect of any serious fall in the prices of butter and cheese below current levels, by means of a price-insurance plan under which Exchequer assistance will be payable, on a prescribed scale, to the Boards in respect of standard gallonages of milk used in factories for butter and cheese, and milk made into cheese on farms, in the event of the average prices of imported butter and cheese falling, over a period, below 100s. and 56s. per cwt. respectively. The present average prices of imported butter and cheese are of the order of 115s. and 72s. per cwt. respectively. The corresponding prices in February, 1934, when the Milk Act proposals were announced, were 71s. 6d. and 47s. 6d. per cwt. respectively.

(8) The advances to the Milk Marketing Boards of Great Britain under the Milk Act, 1934, and subsequent legislation, involve the Boards in a contingent liability during a period ending September 30th, 1940, to repay to the Exchequer an amount estimated at over £3,400,000. This liability only arises if cheese or butter prices rise to a certain level, and it is anticipated that the repayments which will have accrued by October 1st, 1937, will amount to approximately £150,000. It is proposed to release the Milk Boards from any balance of this liability accruing after September 30th, 1937.

(9) In order to give the necessary momentum to the production of quality milks in Great Britain, the Government propose that Exchequer contributions should be made at diminishing rates, for a period of five years, towards the additional costs incurred in the production of milk of the Accredited standard, milk of the Tuberculin Tested standard and milk from Attested Herds. This assistance will be partly in the form of quality premiums additional to those now receivable by producers, but they will also be, in part, in relief of the contribution towards quality premiums which all producers in England and Wales make to-day under the Milk Marketing Scheme. It is proposed that the rates of the Exchequer contributions will be fixed for five years in the case of the Accredited and Tuberculin Tested schemes but that in the case of the Attested Herds scheme the rates should only be fixed for three years and that milk from attested herds shall be assisted in the fourth and fifth years at rates to be determined following a review of the position during the third year.

*Cmd. 5533. To be purchased directly from H.M. Stationery Office at the following addresses: Adastral House, Kingsway, London, W.C.2; 120, George Street, Edinburgh, 2; 26, York Street, Manchester, 1; 1, St. Andrew's Crescent, Cardiff; 80, Chichester Street, Belfast; or through any bookseller. Price 2d. net.

†An announcement on this subject was made by the Financial Secretary to the Treasury in reply to a Parliamentary question on July 28th, 1937.

however, they still found albumin in the urine and the specific gravity had gone up. It was very difficult to explain such cases unless one had got a definite leak with nephrosis. He thought that might be a possible explanation.

(10) The premium on Accredited milk sold by wholesale now payable from the pool funds of the Milk Marketing Board in England and Wales is at the rate of 1d. a gallon, the whole cost of which is borne by the pool; it is proposed that, for the five-year period beginning on October 1st, 1938, this should be increased to 1¼d. a gallon, of which the Exchequer contribution would be ¾d. for each of the first two years, ½d. for each of the third and fourth years and ¼d. for the fifth year, the balance each year to be borne by the pool. The proposed rate of premium of 1¼d. per gallon should encourage a large and increasing number of producers to take up the production of milk of this standard.

(11) As provided in the amendments of the English Milk Marketing Scheme which Parliament has recently approved, the Milk Marketing Board will pay a special quality premium on sales of Tuberculin Tested milk sold by wholesale, which will no longer be exempt from the Scheme. For the first two years of the five-year plan under these proposals, the premium will be at the rate of 1d. a gallon, towards which the Government propose that the Exchequer should contribute ½d. For each of the third, fourth and fifth years, it is proposed that the Exchequer shall contribute ¼d. a gallon and that the premium shall not be less than ¾d. a gallon.

(12) Under the Agriculture Bill of this Session, financial assistance is provided to encourage the eradication of bovine tuberculosis by means of the Attested Herd Scheme. This assistance includes an Exchequer payment of 1d. a gallon on milk from Attested Herds, the Milk Boards being under a contingent obligation to assume this liability in due course. In order to accelerate the flow of pure milk under that scheme, it is proposed for each of the first two years that the premium on sales by wholesale should be increased to 2d. a gallon, of which the Exchequer would contribute 1½d. and the pool would be required to contribute ½d. as soon as these proposals came into force. For the third year, it is proposed that the premium that the Boards shall be required to pay shall not be less than 1¾d. a gallon towards which the Exchequer will contribute 1¼d. a gallon. As stated above, the rates of assistance for the fourth and fifth years will be determined later. In view of these new liabilities, it is proposed to release the Boards from the contingent obligation mentioned above.

(13) The following Table shows the rates of premium which the Board will be required to pay in respect of the various categories of quality milks and the Exchequer contributions thereto:—

—	Accredited Milk.		T.T. Milk.		Milk from Attested Herds.	
	Pool Premium.	Exchequer Contribution.	Pool Premium.	Exchequer Contribution.	Pool Premium.	Exchequer Contribution.
	d.	d.	d.	d.	d.	d.
1st year ...	1¼	¾	1	½	2	1½
2nd year ...	1¼	¾	1	½	2	1½
3rd year ...	1¼	½	¾	¼	1¾	1¼
4th year ...	1¼	½	¾	¼	—	—
5th year ...	1¼	¼	¾	¼	—	—

In view of the difficulty of estimating at this stage the effect of these proposals upon the development of the Attested Herds Scheme during the first three years, it is not proposed to fix the respective amounts of Pool Premiums and Exchequer Contributions for more than the period shown. Exchequer assistance will be given for the fourth and fifth years also; it is contemplated, however, that it will be on a diminishing scale, and as the amount can only be determined when the development of the scheme is known, it is proposed to leave this question to be reviewed during the third year in the light of the circumstances of the industry and the working of the scheme.

The producer of Tuberculin Tested milk will thus earn premiums amounting to 1¼d. (accredited premium) plus 1d. (T.T. premium) equals 2¼d. per gallon in the first two years and 1¼d. plus ¾d. equals 2d. in the remaining three years, towards which the Exchequer will contribute:—

	d.
1st year	1¼
2nd year	1¼
3rd year	¾
4th year	¾
5th year	½

Similarly the producer of Accredited Milk with an Attested Herd will earn premiums amounting, in the first two years to 3¼d. per gallon, and in the third year to 3d. per gallon towards which the Exchequer will contribute:—

	d.
1st year	2¼
2nd year	2¼
3rd year	1¾

(14) The position of producer-retailers in England and Wales has been improved by the recent amendments to the Milk Marketing Scheme which have had the effect of placing their contributions on a new basis. In view of this, some modification of these proposals will be proposed in their case.

(15) It is proposed to make corresponding provisions in relation to the production of quality milks in Scotland, but certain adjustments may be necessary to meet the special Scottish conditions.

(16) The Committee on Animal Diseases of the Economic Advisory Council recommended that the introduction of some measure of compulsory pasteurization should no longer be deferred and the Government propose that, subject to certain conditions, any local authority shall be enabled to apply to the Minister of Health, or the Secretary of State for Scotland, for an Order making compulsory the efficient pasteurization of milk sold by retail in its area. These conditions are that no such Order shall come into operation within two years after it is made, that milk from tuberculin-tested herds and sterilised milk shall be exempt from its scope and that milk retailed from dairies where the milk is derived from a single herd shall be exempt for three years from the date of operation of the Order.

(17) The Reorganisation Commission for Milk, and other bodies that have examined the problem of milk distribution in recent years, have expressed the view that the present methods of distributing milk are, in many respects, capable of improvement. This appears to be generally accepted by those who speak on behalf of the distributive trade. Accordingly, the Government propose, in the legislation that will be necessary to carry out the present proposals, to include provisions to facilitate the improved organisation of the distributive service.

(18) It is proposed also to adopt the machinery of service schemes on the lines of those for which provision is made in the Livestock Industry Act of the present Session of Parliament, so as to enable the various sections of the industry to finance co-operatively services of common interest.

(19) The Government propose that a permanent Milk Commission should set up to keep under review the production, marketing and consumption of milk (including the effect of prices on demand) and to be of general assistance to the industry and to the Ministers concerned. The primary duties of the Commission will be concerned with the improvement of milk distribution and with conciliation and arbitration between the Milk Marketing Boards and the milk buyers in business matters. Apart from this, it is not proposed to disturb the existing arrangements for the negotiation of contract terms and prices or the public safeguards provided, under statute, by the Consumers' Committees and the Committees of Investigation. The Government propose, however, to take steps to enable the views of consumers to be made available to the Milk Marketing Boards at an early stage in the contract negotiations.

The Commission will be required to present an annual report for submission to Parliament.

(20) The Government regard the continuance of the scheme for the provision of cheap milk for school children as an essential feature of their proposals and they are confident that in this matter the co-operation of the industry will, as at present, be forthcoming. This scheme has now proved its merits, and has resulted in a large increase in the consumption of milk in schools. In making arrangements for its continuance, the Government will consider the need for minor modifications to meet difficulties which have arisen in its administration.

(21) The Government attach not less importance to an increased consumption of milk by expectant and nursing mothers, and children under school age, and they will bring forward proposals for securing, in co-operation with the industry, a reduction in the price of liquid milk to local authorities for the purpose of their maternity and child welfare arrangements. Local authorities will thus be in a position to extend their present schemes under which milk is made available either free or at a reduced price as circumstances may require.

(22) The Government commend the proposals as a whole as a policy which will provide for the dairying industry a basis for an increasing sale of milk in the liquid market, will secure cheap milk for those classes of consumers to whom it will be of particular value and will provide for consumers generally a permanent improvement in the cleanliness and purity of the milk supply. The proposals should lead to a reduction in the cost of distribution and, as the eradication of disease among dairy herds progresses, in the cost of production; they should lead to the enhancement of technical efficiency; and they will be a notable step towards the ultimate objective which must be to ensure that only clean and pure milk is offered for sale for liquid consumption.

Legislation to give effect to these proposals will be introduced as early as practicable next Session.

(23) The Ministers concerned have deferred consideration of a scheme, submitted under the Agricultural Marketing Acts, for regulating the marketing of milk products. Subject to the wishes of the promoters in the light of these proposals, the Ministers concerned will be prepared to give this scheme further consideration.

(24) The following statement shows the estimated cost of the Exchequer contributions to the Quality Premiums in each of the next five years:—

Year *Oct.-Sept.*	*Exchequer Contributions to Quality Premiums (excluding expenditure under Part IV of the Agriculture Bill).*
	£
1938-39	1,700,000
1939-40	1,962,000
1940-41	1,781,000
1941-42	1,425,000*
1942-43	800,000*

*Does not include assistance for Attested Herds for the fourth and fifth years—*see* paragraph 13.

The Veterinary Record

September 18th, 1937.

The Milk Policy of the Government

THE issue of a pamphlet, reproduced in this issue, giving in detail the projected milk policy of the Government allows the profession to consider this policy carefully. Little of a controversial nature is contained in the pamphlet, and there is little doubt that the policy outlined will receive the support of the dairying industry.

The Government has rightly emphasised at the outset that "the only sure foundation for the prosperity of the industry is an increased consumption of liquid milk. An essential step towards this objective is to establish public confidence in the cleanliness and purity of the milk supply, and thereby to quicken demand for what is one of the most valuable of human foods."

To this end it is proposed to assist the farmer materially in his efforts to improve the quality of his milk supply. Grants are to be made by the Exchequer towards quality milks, in addition to those made by the Milk Marketing Board, and the amounts of these grants have been wisely fixed for a period of five years for Accredited and T.T. milk, and for a period of three years with a promise of extension to five,

for milk from Attested Herds. The details of these grants are given elsewhere in this issue, but it may be mentioned here that for the first year, producers will receive 1¼d. per gallon for Accredited milk, 2¼d. (1¼ + 1d.) per gallon for T.T. milk, and 3¼d. (1¼d. + 2d.) per gallon for milk from Attested Herds. The Government thus clearly illustrates its intention to assist the improvement of the quality of our herds, and of the milk they produce.

In order to develop still further the confidence of the consuming public in the milk supply, the Government has wisely accepted the view that the policies of herd improvement, and of pasteurization of milk are not contradictory, but are, in fact, collateral. The Government "propose that, subject to certain conditions, any local authority shall be enabled to apply to the Minister of Health, or the Secretary of State for Scotland, for an Order making compulsory the efficient pasteurization of milk sold by retail in its area. These conditions are that no such Order shall come into operation within two years after it is made, that milk from tuberculin-tested herds and sterilised milk shall be exempt from its scope and that milk retailed from dairies where the milk is derived from a single herd shall be exempt for three years from the date of operation of the Order." We are glad to note that the Government has laid emphasis upon *efficient pasteurization* and presumably, therefore, the local authorities will be expected to insist upon this efficiency in their areas.

The Government also intends to set up a permanent Milk Commission "to keep under review the production, marketing and consumption of milk (including the effect of prices on demand) and to be of general assistance to the industry and to the Ministers concerned." We may hope that this Commission will take steps to get adequate advice upon those sections of its work which concern the veterinary profession.

In conclusion, we may express our pleasure at the determination of the Government to help to improve the quality and safety of our milk supply, and we may rest assured that the veterinary profession, by encouraging farmers in their efforts, will contribute substantially to the success of these schemes.

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Scientific Criticism

MANY contend that to-day much is published in scientific periodicals that would probably be withheld from publication if the author considered that there was any likelihood of his work being submitted to severe criticism. The mass of scientific literature being published lends itself to the charge that many accounts of work hastily finished or superficially done are unnecessarily included in these journals. In an endeavour to ensure that only work of sufficient merit is published, most editorial boards—including our own—submit papers received from contributors to one or more specialist referees as a routine procedure. The referees make whatever criticism of the paper they deem necessary, and this criticism, if any, is dealt with confidentially by the editorial board and the contributor, in such a way that the paper may be suitably amended before publication. By such a genuine, but amicable, criticism, both authors and journals benefit.

Occasionally papers are published which merit severe criticism, and a suitable reply, giving this criticism, may be written by another writer. In these instances, the published criticism is usually wisely restricted to a discussion of the scientific data given in the first paper, these data being reviewed objectively and in a manner devoid of all personal feeling. If this procedure is followed, everybody again benefits and personal feelings are left unruffled.

An unusual method of criticism, which has little to be said in its favour, consists more of an attack fraught with sarcasm, upon the scientific reputation of an author, than of a purely critical consideration of the results that the author has actually produced. This method of criticism is of little value to the scientist, and, in fact, usually defeats its own end, for the element of personal criticism is apt to distract the reader's mind from whatever critical value there may be in the article.

We regret that such criticism as this has lately been made elsewhere by an eminent member of our profession, upon one of his colleagues. We are not discussing here the merits or demerits of the original paper criticised; these, no doubt, will receive full treatment in other journals, but we wish to register an emphatic protest against the use of subjective criticism in any review or article which should be devoted to a discussion of scientific facts or theories in a cold, objective manner.

ERADICATION OF TUBERCULOSIS IN CATTLE

The schemes for eradicating tuberculosis from individual herds of cattle, which were instituted respectively by the Ministry of Agriculture and Fisheries and the Department of Agriculture for Scotland in 1935 under the title of Attested Herds Schemes, are now making marked progress. This is evident from a comparison of the Register of Attested Herds just issued, which shows the position on July 31st, with that published six months earlier.

In England and Wales there were at the end of July, 346 attested herds comprising 13,870 cattle, as against 208 herds (7,944 cattle) on January 31st. The corresponding figures for Scotland are 452 herds comprising 36,725 cattle compared with 256 herds (21,025 animals) six months ago. A large number of applications for Certificates of Attestation in respect of other herds are also under consideration.

The scheme for England and Wales has recently been amended (as was the scheme for Scotland last year) so that not only is the herd owner encouraged to attest his herd by the prospect of receiving a bonus on the milk which he sells through the Milk Marketing Scheme, but he may receive assistance in the process of eradicating tuberculosis from the herd. Instead of, as formerly, being left to complete the eradication entirely at his own expense, he can, as soon as he has reduced the number of reactors in the herd to about 10 per cent., avail himself of that part of the scheme which provides a substantial contribution towards the cost of further tests. The value of this monetary assistance has been realised by many herd owners and an impetus has thus been given to the eradication of disease which will probably bear fruit in due course in a large increase in the number of herds attested.

A Certificate of Attestation is virtually a guarantee of the freedom of a herd from tuberculosis, a fact which is of great importance to those who desire to obtain disease-free cattle. The Register just issued gives the addresses of the owners of attested herds and particulars of the breeds constituting the herds. Copies of the Register and particulars of the schemes may be obtained from the Ministry of Agriculture and Fisheries and the Department of Agriculture for Scotland.

CLINICAL COMMUNICATION

Hemlock Poisoning in Horses

HUGH MACDONALD, M.R.C.V.S.
EXETER

In June, 1936, two cases of poisoning of horses by hemlock (*Conium maculatum*) were observed, and one further case, again in a horse, was met with last June.

In the first two cases both horses were affected almost simultaneously. The horses had been working as a pair in the hay field, and for a period of half an hour were unharnessed and allowed to graze on a piece of waste land close at hand. One hour after restarting work one of the horses stumbled and fell, and when the harness was loosened it unfortunately rolled into a ditch some four feet deep.

When seen by the writer, the horse was still in the ditch and appeared to be unconscious. The other horse was used to drag it out, but five minutes after this was accomplished the second horse suddenly fell forward without a struggle and remained quiet on the ground.

The symptoms shown by both horses were similar—loss of sensibility; corneal reflexes present; breathing, pulse and temperature normal, and very little trembling.

Strychnine sulph. ½ gr., was given to each horse, followed in 15 minutes by ammon. carb. 2 oz. in a gallon of water, given by stomach tube. Twenty minutes later the first horse showed signs of revival, and after two attempts at rising it finally did so, and remained quite steady on its feet. Fifteen minutes later the other horse got to its feet. Both animals then walked home, a distance of some two miles, without any apparent evidence of illness. No further treatment was necessary.

The third case observed showed more symptoms of the classical type, in that there was trembling before going down, and that the breathing was slightly accelerated while the animal was down. This horse remained more or less unconscious for two hours; and after it got up it appeared normal for ten minutes, at the end of which time slight trembling commenced in the hind quarters. This very rapidly increased in intensity and extent until the whole body quivered to such a degree that control was lost and the horse fell forward on its head. The trembling gradually ceased in a few minutes.

The same treatment was adopted as in the two previous cases, except that the ammon. carb. was not given until the relapse occurred. After about an hour the horse got to its feet

and walked quietly to a loose-box, without further relapse.

The horses in question were of the Shire type.

Hemlock was found in each instance, and there was evidence that a quantity of the flowers and leaves had been eaten by the horses. In the first two cases the animals had had the opportunity to eat the plant for half an hour only, and in the other case the horse just picked some of the leaves and flowers at each turn as it was mowing the small field.

It is interesting to note that there was no dyspnoea present, and that in two instances there was no warning of approaching paralysis until the animals actually stumbled and fell.

Hemlock is so liberally distributed over the country that one would expect more instances of poisoning from the ingestion of the plant, although it is possible that animals eat it only in the absence of other food.

ABSTRACTS

[Histopathology of Natural and Experimental Canine Distemper. DE MONTREUX, W. A. (1937.) *Amer. J. Path.* **13.** 75. 187-212.]

Post-mortem histological examination of the organs in 13 fatal cases of distemper as compared with 18 normal dogs, showed the presence of cell-inclusions which the author considers to be characteristic of the disease. Fifty-three susceptible puppies inoculated with various strains of distemper virus showed similar *post-mortem* findings.

The characteristic lesions occurred mainly in lungs, spleen and mesenteric lymph glands. Nuclear and cytoplasmic inclusions were found in both bronchial and alveolar lung cells. The cytoplasmic inclusions were well-defined, round or oval, vacuolated, acidophilic bodies lying between the nucleus and outer margin of the cell. Often two or three were found in a single cell. The nuclear inclusions were non-vacuolated, variable in size and irregular in outline. Similar inclusions were observed in the lungs in experimental cases only when the virus was introduced by way of the respiratory tract, and the constancy of their occurrence in the natural cases studied pointed to the common portal of infection being the respiratory tract.

In the spleen in both natural and experimental disease there was proliferation of reticulo-endothelial cells, with nuclear inclusions in some of those cells, usually within the follicles. The same type of inclusion body was noted in the reticulo-endothelial cells of the mesenteric lymph glands. In the majority of cases the bile-duct epithelium showed similar inclusions. In cases which showed nervous symptoms perivascular accumulations of endothelial and mononuclear cells were found in brain tissue and nuclear and cytoplasmic inclusions could be demonstrated in both neurons and glial cells. In many cases the cytoplasmic inclusions appeared indistinguishable from Negri bodies, but the presence of associated nuclear inclusions offered a basis of differentiation.

An acute encephalitic type of distemper is described as occurring in two five-weeks-old puppies whose mother had had a non-fatal attack of the disease about ten days previously. The animals died after an illness of about 30 hours and none of the usual clinical signs of distemper was present. At the onset of the disease the puppies were extremely nervous and refused all food and water. They rapidly weakened, became lethargic and died in coma. The characteristic lesions described above were well marked and the presence of the distemper virus was confirmed by animal inoculation. A similar type of disease developed in a number of puppies given fairly large doses of virus by intraperitoneal, intravenous and intracerebral routes.

Similar histological findings to the above are recorded in experimental infections of ferrets.

A. R.

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[Pneumonia in Foals due to Corynebacterium equi. RAJAGOPALAN, V. R. (1937.) *Ind. J. Vet. Sci.* **7.** 1. 38-53.]

In the author's opinion an appreciable number of the non-specific diseases of foals under twelve weeks old, and more especially those associated with the respiratory tract, are due to *C. equi*, a pigmented diphtheroid. This has been described by workers in Europe, Australia and America but not previously in India.

The records of two breeding establishments in the Punjab are discussed with the help of tables and photographs.

The incidence was heavier during the summer months and 60 per cent. of the foals died between the first and third months after birth.

An attack is heralded by a subnormal temperature, rising in a few days to 105° F., and there is a watery discharge from the eyes and nostrils, laryngitis and a dry cough. Frequently swelling of the joints occurs and typical pneumonic symptoms develop. Emaciation is rapid and the foal dies in eight to ten days after having contracted the disease.

At *post-mortem* examination the most striking lesion is that of abscess formation in the lungs and mediastinal glands, these containing inspissated pus or fluid and having a characteristic foul smell.

The bacteriological aspect of the organism is dealt with fully.

Though a treatment consisting of an intravenous injection of 25 c.c. of 1 per cent. mercurochrome, cough electuary and drenching with tinct. iodidi. seemed to be beneficial, great stress is laid on good hygienic measures, keeping mares and foals out all the year round and practising a paddock rotation.

N. S. B.

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[**Nerve Suture.** Lloyd, G. (1936.) *J. Roy. Army Vety. Corps.* **7.** 3. 121-137.]

This article deals with the operation of nerve suture in relation to the relief of roaring in horses, namely, the suturing of the left recurrent laryngeal nerve either to the common trunk of the vagus and sympathetic, to the spinal accessory or to the phrenic, with the object of bringing about the re-enervation of the paralysed member. The author reviews the historical aspect of the problem in considerable detail, particularly the work of Sir Frederick Smith. He then gives details of four operations on roarers carried out by himself during the years 1933-'34 in which the distal end of the severed trunk of the left recurrent laryngeal nerve was sutured to the phrenic. Some degree of improvement in the roaring condition was effected in two of the cases. Two died of post-operative complications attributable to the lengthy anaesthetisation. The author concludes that the operation, though a practical proposition, is only likely to prove of value when it is carried out in the initial stages of degeneration of the recurrent laryngeal nerve; that is to say, prior to the onset of fibrous changes in the crico-arytenoideus posticus muscle.

C. D.

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[**Multiple-Lesion Enzootic of Lactating Ewes.** Weinberg, M., Forgeot, P., and Maureu, A. (1937.) *Bull. Acad. vét. France.* **10.** 2. 117-123.]

The authors describe an enzootic among lactating ewes in which 18 animals out of a flock of 150 fell ill and of these 18, twelve died.

The condition at first appeared to be a simple mammitis and diplococci were isolated from the gland, but it was felt that these could not explain the lesions seen in liver, spleen, kidneys and suprarenal capsules.

Since it was found that the feeding was bad it was decided to examine further the possibility of the existence of infectious enterotoxaemia.

Bacteriological examination of the small bowel contents failed to show the presence of *Clostridium ovitoxicum* and filtrates proved innocuous to mice when given in large doses subcutaneously.

From the liver and bone marrow, on the other hand, there were isolated anaerobically a coliform organism and a Gram-positive rod resembling *Clostridium welchii* (*perfringens*). The former proved harmless to the guinea-pig, while the latter killed in 24 hours following a dose of 1/20 c.c. culture intramuscularly. *Post-mortem* examination of the guinea-pig showed the usual lesions caused by *Cl. welchii* and culture revealed the presence of the organism in the oedematous liquid of lesions and in the heart blood. Anti-welchii serum gave complete protection against the organism.

On fuller enquiry into the condition as it occurred in nature it was found that the first symptoms noted by the shepherd were that ewes lost their appetites and went lame. Closer observation revealed a rise of temperature up to 40·3° C. and swelling of one side of the mammary gland stretching from the lips of the vulva nearly to the forelegs. Three days was the average duration of illness and just before the end the animals' temperature fell to about 36·2° C. It may be noted that no crepitation could be detected in the local lesion.

From a bacteriological point of view the condition is interesting since it provided exceedingly virulent strains of *Cl. welchii*; one, indeed, was the most virulent strain of *Cl. welchii* of either human or animal origin in the authors' laboratory.

J. G. M.

* * * * *

[**A Study of Blackleg and its Complications.** Breed, F. (1937.) *J. Amer. Vet. Med. Ass.* **43.** 4. 521-528.]

From a study of "blackleg" in America the author comes to the conclusion that while *Clostridium chauvoei* is the true cause of uncomplicated "blackleg," many cases of clinical "blackleg" show also the presence of *Cl. septique* and some few also *Cl. novyi* (*oedematiens*).

Microscopic examination of material from "blackleg" cases does not provide a diagnosis of the type of infection and further laboratory methods must be employed.

In some districts it was observed that the usual "blackleg" vaccine prepared from *Cl. chauvoei* was not giving the excellent results obtained in other areas and it was found that in these areas the infection was often of the complicated type.

Some young cattle were inoculated with a chauvoei-septique vaccine in these areas and grazed on pastures which were known to have an exceptionally bad reputation for "blackleg," deaths even following inoculation with the simple *Cl. chauvoei* vaccine. There was no loss and while no controls were included the author thinks that there is justification for his view that the mixed chauvoei-septique vaccine is

REVIEW

[**"Elements of Stabling."** By Captain R. M. S. Barton. 126 pages; 31 illustrations from photographs.]

This is a book that may be profitably studied by every man who contemplates the purchase of his first horse. If his means make it necessary for him to do most of his own stable work, he will here find valuable advice which, if intelligently followed, will save him from endless trouble and expense. On the other hand, should he be able to afford a groom, Captain Barton's book will enable him to meet his employee on more equal terms and to check the man's work, a great advantage and one that is cheaply purchased by a few hours' attentive reading.

To the veterinary student this book should be both profitable and interesting. In these days, unfortunately, many students enter College without any practical experience of dealing with horses; comparatively few have ever ridden a horse, many have never even touched one. To these unfortunates experience often comes slowly, bringing many humiliations in its wake. Nothing can be more crushing than the polite scorn of the old groom or horse-keeper, when an unhappy student, seeing practice perhaps for the first time in a country district, makes some appalling blunder in elementary stable-science. From such painful experiences a student can, in great measure, protect himself by reading, and in "Elements of Stabling" he will find much useful matter, clearly stated. Captain Barton is very sound without being dogmatic. In this he avoids an error too common in works of this description, namely, an irritating insistence that only the one method described is, or can be, correct. The author, on the contrary, repeatedly gives alternative ways of treating horses, and remarks with admirable common sense, that, as animals differ from each other, so one rule will not hold good for all: a rare breadth of view and one to be recommended to many writers.

This is not the only point on which the author of "Elements of Stabling" has shown a commendable restraint. He has resisted the temptation to devote a large part of his book to quack remedies. The passion for amateur doctoring is one of the commonest and most deeply-rooted in human nature, and we fear that Captain Barton may have sacrificed some measure of popularity to his sense of probity, but such sacrifice has added to the true value of his work.

At the commencement of his admirable chapter headed, "Preventions—Medicines—Cures," he earnestly recommends calling in veterinary advice wherever it is possible to do so. He goes on to describe some first-aid measures, so excellent and necessary that every horse-owner should learn them by heart. He also gives a few simple remedies for troubles that cannot fail of diagnosis even by the tyro.

And it is for the tyro Captain Barton writes, a fact he has never allowed himself to forget. In this lies much of the usefulness of his work, for it is rare to find an author who, himself so well-informed, has yet patience for the elementary details of his subject. Too many would-be instructors forget that even words of one syllable are difficult for the unfortunate who is ignorant of his letters.

A useful and reliable work, well and clearly written.

superior to the simple chauvoei vaccine for use in these areas.

Experimentally the author showed in lambs that simple chauvoei vaccine gave adequate protection against pure chauvoei infection but not against infections of septique or of septique combined with chauvoei.

J. G. M.

REPORTS

Southern Rhodesia

REPORT OF THE DIRECTOR OF VETERINARY RESEARCH FOR THE YEAR 1936

The work of the department was hampered during 1936 owing to the fact that for half that period only one professional officer was employed. In a young country like Southern Rhodesia, where the livestock industry is of paramount importance and where there is a clamant need for the investigation of local veterinary problems, the loss of even one veterinary surgeon transferred to another department, as appears to have happened in the present instance, means that a good deal of important work had to be left over for another year and investigation of animal disease "on the spot" had to be given up for the more pressing routine work. All the same, progress to a considerable extent has been made, particularly on foot-and-mouth disease and theileriasis.

Early in the year the Director demonstrated the intramucosal inoculation of the tongue and we assume that in future outbreaks this method, which has been practised so successfully in adjoining territories, will be employed. The inoculum is composed of vesicular fluid suspended in glycerine saline and the method has the advantage of supplying sufficient inoculum with reliability, rapidity and ease for the large

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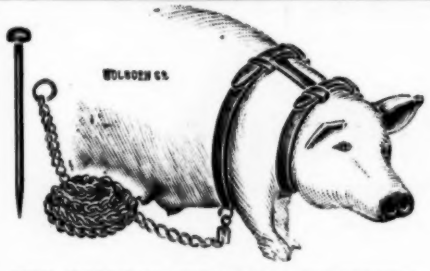

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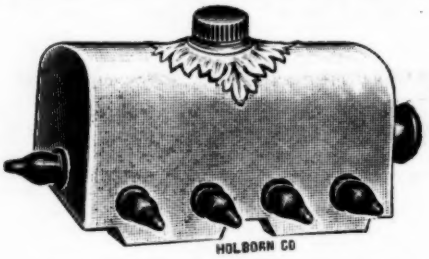

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numbers of animals one has to deal with under South African conditions.

The intranasal inoculation introduced by Bevan served its purpose well when the disease first entered the territory, but the intramucosal method is preferable because of its certainty of producing the requisite quantity of inoculum of definite infectivity with regularity. This improved inoculation is a tangible and practical result of research work on foot-and-mouth disease, to the necessity for which under South African conditions we drew attention in a recent review.

There is an interesting observation in the course of the report on the demonstration of the *Rickettsia ruminatium* in cases of heartwater. While intravenous inoculation of susceptible animals with blood from affected animals has proved positive there has often been great difficulty in demonstrating the Rickettsia in the blood stream. The Director has found that smears taken from the intima of the jugular vein of affected animals contained perfect specimens of *Rickettsia ruminatium* in large numbers, a factor which may have an important diagnostic value in the laboratory.

Cases of theileriasis have occurred with marked frequency during the year and the Director has been occupied in investigating cases caused by *Th. parva* and *Th. mutans*, and also deaths due to theileriasis which conformed to no known type. The symptoms were closely allied to those of East Coast fever and at first it was suggested that it was a per-acute form of that disease. Later cases which ended in death or recovery after a period of a fortnight rendered this suggestion untenable.

Post-mortem examinations failed to show typical lesions and the infection of erythrocytes was absent. Koch's bodies were demonstrated in spleen and gland smears and in some cases were as numerous as those in typical East Coast fever. In many cases Koch's bodies were present in blood smears, but as a rule not a single piroplasm could be found. Repeated attempts to transmit the disease by inoculation were unsuccessful and here the Director describes what he terms the "pin-cushion method" of inoculation, by which injections are made simultaneously subcutaneously, intravenously, intradermally and intraglandularly, and occasionally intraperitoneally also.

What association there is between these newly discovered cases of theileriasis and East Coast fever has yet to be determined and we shall await with interest further reports which, it is indicated, will soon be published. It is pointed out that until definite information is available these obscure cases of theileriasis must be treated as East Coast fever.

The usual laboratory products and service have been forthcoming and much of the vaccine which the Onderstepoort laboratory so generously supplies to adjoining territories is now being manufactured at the Salisbury laboratory. As much as 55,848 doses of quarter-evil vaccine were manufactured locally and issued. Infectious abortion vaccine, however, with an issue of 280 doses only, showed a decrease on previous years.

The neurotropic horse sickness vaccine which was first used in the territory in 1934, appears to have been in great demand, as 1,046 equines were immunised. This is a considerable increase on previous years and indicates the faith the public have in this improved method.

In Southern Rhodesia there is urgent need and great scope for the veterinary research worker, and we hope the Director will be given the additional assistance to which we think he is entitled.

* * * * *

City of Manchester

SUMMARY OF THE ANNUAL REPORT OF THE MARKETS COMMITTEE FOR THE YEAR ENDED MARCH 31st, 1937

This comprehensive report, prepared by the General Manager of the Markets Department, includes work carried out by the veterinary staff in connection with meat inspection and the Diseases of Animals Acts.

During the year to March, 1937, 74,560 cattle, 435,204 sheep and lambs, 11,553 calves and 12,586 pigs were slaughtered at the city abattoirs. These figures show a decrease of 2,679 in the number of cattle and 1,630 in the number of pigs compared with the preceding year. On the other hand, an increase in the killing of sheep and calves is recorded by the figures of 23,740 and 925 respectively. The number of carcases of beef exposed for sale in the wholesale market and derived from animals killed elsewhere in the British Isles shows an increase of 1,107 on 1936 figures, and sheep carcases a decrease of 5,198 for the same period. The numbers of imported carcases exposed for sale were: beef, 48,054; mutton and lamb, 574,484; veal, 201; pork, 11,902. The percentage of home-killed and imported meat works out at 52·8 and 47·2 respectively, which shows a slight increase in the home-killed figure over the preceding year.

Meat Inspection.—The numbers of carcases of animals slaughtered at the city abattoirs and which were condemned were as follows: beef, 636; mutton, 545; veal, 45; pork, 45. These figures form an interesting comparison with the numbers of condemned dressed carcases consigned from places outside the city, which were: beef, 828; mutton, 180; veal, 364; pork, 355. The conclusion one draws from these

figures, after allowing for the inevitable loss due to decomposition and damage, is that a very considerable amount of meat has access to the market which has not been efficiently inspected at its source. It is to be noted that one in every 8·5 carcases of beef and one in every 48·5 carcases of mutton received in the markets from other places in the British Isles were condemned. Tuberculosis accounted for the condemnation of 563,765 lb. of meat, this figure including 836 whole carcases of beef, 28 carcases of veal and 127 carcases of pork. In addition, 260 lb. of meat were condemned on account of anthrax. Magistrates' orders to the number of 13 were requested for the destruction of unsound food. Three prosecutions under the Public Health Acts, 1875 and 1890, resulted in fines totalling £182 being imposed.

Diseases of Animals Acts.—*Foot-and-Mouth Disease.*—During the year Manchester was within a foot-and-mouth disease infected area for a period of two weeks. *Parasitic Mange.*—There were four outbreaks of this disease, affecting four animals with 104 "in contacts." *Tuberculosis.*—Seven cases were recorded, but no reference is made to the type of tuberculosis encountered. *Swine Fever.*—Two outbreaks are noted, causing the death of one pig and the destruction of 20 "in contacts." This is a comparatively small number of outbreaks in view of the extent of the disease elsewhere. *Anthrax.*—Two cases of this disease were observed during the year, and presumably a carcase or part-carcase affected gained access to the meat market.

ROYAL (DICK) VETERINARY COLLEGE

EXTENSION

Plans for the extension scheme at the Royal (Dick) Veterinary College, Edinburgh, were passed by the Edinburgh Dean of Guild Court on September 3rd. When completed the new building will include improved accommodation for the departments of anatomy, hygiene, epizootiology, and additions to the clinical and X-ray departments, with laboratories, lecture rooms, and demonstration theatres, research, and animal rooms. The site is immediately to the north of the existing college. The whole scheme is estimated to cost £40,000, and will be proceeded with in sections as finances permit. The architects are Messrs. Lorimer and Matthew.

* * * * *

ARMY VETERINARY SERVICE

London Gazette—War Office—Regular Army

Sept. 14th.—Maj. G. F. Watkins is secd. on appt. to the Ind. Continuous Serv. Cadre (Aug. 18th).

Notes and News

The Editor will be glad to receive items of professional interest for inclusion in these columns.

Diary of Events

Sept. 24th.—Meeting of Southern Counties Division, N.V.M.A., at Winchester, 2.30 p.m.

Sept. 25th.—Annual General Meeting, Society of Veterinary Practitioners (Division, N.V.M.A.) Grand Hotel, Birmingham, 3 p.m.

Sept. 27th.—Meeting of the Editorial Committee, N.V.M.A., at 36, Gordon Square, W.C.1, 4 p.m.

Sept. 27th.—Royal Veterinary College: Formal Opening of new Session at Camden Town. Lieut.-Colonel P. J. Simpson, D.S.O., T.D., J.P., F.R.C.V.S., to attend as President of the College.

Oct. 1st.—R.A.V.C. Old Comrades' Association: Annual Dinner, Anderton's Hotel, Fleet St., E.C.4; 7.30 p.m.

Oct. 4th.—N.V.M.A. Committee Meetings.

Oct. 5th.—N.V.M.A. Council and Committee Meetings.

Oct. 6th.—R.C.V.S. Committee Meetings.

Oct. 6th.—Victoria Veterinary Benevolent Fund Council Meeting.

Oct. 7th.—R.C.V.S. Committee Meetings.

Oct. 8th.—R.C.V.S. Council and Committee Meetings.

Nov. 9th.—Royal Veterinary College: Opening of new buildings at Camden Town by His Majesty King George VI.

* * * * *

OBITUARY

Professor Oluf Bang

It is with regret that we have to announce the passing, at the early age of 56, of Professor Oluf Bang, of Copenhagen. His death occurred suddenly in August while he was on holiday in Norway. Oluf Bang, the son of the late distinguished Professor Bernhard Bang, was a familiar figure at international congresses connected with veterinary science or agriculture and he was, therefore, like his father, known personally to many British veterinarians. In the international sphere he was assisted, as are so many Scandinavians, by wide linguistic abilities.

Oluf Bang's professional career was a distinguished one. He entered the University of Copenhagen in 1899 with the intention of studying medical science but later transferred his

activities to veterinary medicine, graduating in 1905. From then until 1913 he held various assistantships in the Departments of Surgery and Medicine at the Royal Veterinary and Agricultural College, finally becoming in 1914 Professor in Special Pathology and Chief of the Medical Clinic.

Bang carried out a good deal of original research and the results are published in a number of papers. His first work with Ellermann was a demonstration that a leukaemia of fowls is due to a filterable virus. Still more widely known is his work on tuberculosis, Johne's disease and contagious abortion in cows. Before it had been shown that the Johne's bacillus could be cultivated on appropriate media, Bang was occupied with the question of latency in this disease and he was the first to point out that avian tuberculin could be used for diagnosis. His researches on tuberculosis were concerned with its bacteriology, the uses of tuberculin and of the complement-fixation test in diagnosis, the use of sanocrysin in treatment, tuberculosis in mammals due to the avian type of bacillus, and the pathological anatomy of avian tuberculosis; more recently he was studying the uses of B C G. In connection with contagious abortion, he collaborated with a number of other persons in studies on latency of udder infection, the possible importance of the skin as a portal for the entry of the organism, the uses of the live vaccine in prevention. His activities in the clinical field included investigations on "heart block" in horses, while there should be mentioned finally his work with others on the mineral metabolism of horses and cattle.

With the death of Oluf Bang, the Danish veterinary profession has lost one of its leading men, and one who should normally have had a number of years of active life before him.

. . . . .

David Robert Stoker.—Graduates of the Glasgow Veterinary College will regret to learn of the sudden death, on the 8th instant, in his 57th year, of Mr. Stoker, Janitor of their Alma Mater. He will be affectionately remembered by graduates, students and members of the College staff with whom he came in contact during his eight years of faithful service at the College. A Glasgow graduate writes: "It was fitting that members of the staff, representatives of the S.R.C. and many students accompanied his remains to Rosebank Cemetery, Edinburgh. It is felt that many a kindly thought will be wafted over wide distances by many of us who found in him a friend, and it is hoped that these thoughts will comfort Mrs. Stoker and family in their bereavement."

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PERSONAL

Appointment.—Mr. R. E. Hirtes, M.R.C.V.S., has been appointed Assistant Veterinary Officer to the Sheffield City Council.

Marriages.—McLaren—Borthwick. At North Morningside Church, Edinburgh, on September 8th, 1937, by the Rev. James S. Stewart, B.D., George Good McLaren, M.R.C.V.S., younger son of Mr. and Mrs. Hugh McLaren, "Akra," Boydfield Avenue, Prestwick, to Muriel Netta Allan, elder daughter of Mr. and Mrs. A. M. Borthwick, "Antrim," 27, Ulster Crescent, Edinburgh.

Quinlan—Le Lacheur. On September 11th, at Chichester, Maurice, elder son of Mr. and Mrs. T. Quinlan, of Cork, to Mary Joyce, elder daughter of the late W. J. Le Lacheur and Mrs. W. J. Le Lacheur, of Littlehampton.

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On Leave.—The following officers in the Colonial Veterinary Service arrived in this country, during August, on leave: Messrs. W. Fotheringham (Kenya), W. W. Henderson and T. F. O'Brien (Nigeria).

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AIR-RAID PRECAUTIONS

A course of lectures on Air-Raid Precautions will be held at King Edward's Memorial Hospital, Ealing, on Friday evenings at 8.30 p.m., from September 24th to October 29th.

Any veterinary surgeon who wishes to attend the course should apply to the Hon. Secretary, West Middlesex Division B.M.A., Roundabout House, Greenford, Middlesex.

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FOOT-AND-MOUTH DISEASE

After a period of immunity of about three years a serious epidemic of foot-and-mouth disease has invaded Holland from Belgium and France. The first cases were reported near the Belgian frontier, and district after district was affected until cattle in a great part of the country are now suffering from the disease. In one of the Polder districts there are 400 cases. A cattle show has been cancelled at Leyden and strong measures to cope with the disease have been taken by the authorities.

. . . . .

The existence of foot-and-mouth disease among cattle at Marske, Richmond, Yorkshire, was confirmed on September 12th. The order restricting the movement of animals affects an area lying in the North Riding and in Durham.

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THE BRITISH ASSOCIATION MEETING

(*Continued from page* 1192.)

It was officially announced on Wednesday of last week, at Nottingham, that the Association would meet next year at Cambridge from August 17th to 24th.

On that day the sectional meetings concluded, the most important single event being the discussion, in which six sections participated, on planning the land of Britain, in which Dr. Dudley Stamp, Professor Boswell, Dr. J. S. Huxley, Professor E. J. Salisbury, and Sir Daniel Hall took part.

Three chief points emerged—the need for a detailed survey as a basis for planning; the possibility of increasing the home-grown food supply, and the necessity of planning for amenities. Central responsibility for national

parks was essential. Vested interests, as in grouse moors and deer forests, it was urged, should not be allowed to stand in the way of health, recreation, and the enjoyment of natural beauty for the public. As regards food production we must have a well-defined policy in view. Do we want more food or a greater population employed in agriculture? Are we planning for the urgencies of war or for better nutrition in times of peace?

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Gravity of Erosion in the Empire

Sir John Russell, director of Rothamsted agricultural station, opened a symposium at a joint session of the agriculture and botany sections on the pasture problem.

His general discussion of how to create a satisfactory grass pasture led up to the statement that laying land down to grass increases its fertility and that this was one of the great values of the new fertility policy of the Government in increasing the supply of phosphates and of lime for grass lands. This would increase the growth of the grass, which in turn would increase the fertility of the soil. But if the pasture was broken up there would be stores of fertility remaining in the soil that would survive for some years, an asset which could be cashed for other purposes.

A second point about grass culture was its profound importance to the Empire, especially in the sub-tropical and tropical regions. "Covering land with grass stops erosion," he said. "Some of you may have seen in the *Times* recently an article written by Mrs. Elspeth Huxley on erosion in Africa. That article was in no way overdrawn, for the erosion in the tropical and sub-tropical parts of Africa is very great indeed. I myself am just back from Armenia and the upper hills of Georgia. In that part of Asia there is an enormous amount of erosion, which loads up the streams with silt, wrecks irrigation schemes, destroys the land on the hills, and ruins the land in the valleys. It is one of the most serious problems of modern times.

"One of the most important problems, then, in our own sub-tropical and tropical Empire is to find some way of covering land with rapidly growing vegetation so as to stop erosion. There are some grasses found in East Africa by Dr. Paul Evans, woolly finger grasses, which will grow rapidly, and the use of these is spreading. Also some Australian grasses will grow in dry and hot conditions, and this knowledge is now being applied to the solution of this terrible problem of soil erosion."

Dr. Winifred Brenchley, speaking of the ultimate composition of the herbage from various seed mixtures, described an experiment at Rothamsted in laying down to grass a loam on clay with a number of mixtures sown in parallel strips. Rye grass assumed and retained a dominant position, the Italian variety proving unexpectedly persistent. Cocksfoot established itself easily and maintained its position in varying degrees, but the quantity sown had little influence. Rough-stalked meadow grass was slow in establishing itself, but spread considerably and had invaded areas in which it was not sown. Wild white clover was the only leguminous species that had proved persistent, the quantity in which it occurred being clearly determined by seasonal conditions, especially rainfall.

Problems of Cold Storage

In the section of physics papers were given by Dr. Griffiths and Mr. Awbery, of the National Physical Laboratory, on the contributions of refrigeration to everyday life.

It was clear from the results reported that rapid progress was still being made in this field, and that in the future refrigeration might prove a competitor of the canning industry. Methods are being developed which, on the one hand, cause the minimum of change in the food product, and, on the other hand, permit of easy transport and distribution. The old belief that refrigerated food always showed deterioration in quality and did not taste so good as fresh foods was based on the relatively primitive methods adopted in the early days of the industry.

In America a method of quick freezing, dubbed "imprisoned freshness," was proving very effective with certain categories of meats and fruits. The engineering problems involved in cold storage were formidable. Dr. Griffiths showed a photograph of a South African apple store capable of holding 1,000,000 apples. The girders must be of special construction to take up expansion and contraction; and portholes must be provided for inspection from outside, since to enter the gas-filled interior oxygen apparatus must be used.

Cork, even in special preparations, has now been superseded in insulating power by certain artificial materials, notably expanded ebonite, and, most efficient of all, glass silk. This was made by drawing fine glass threads and spinning them while still hot on a wheel. To illustrate the ease with which this material could be manipulated Dr. Griffiths showed a slide of a woman wearing a glass silk dress. He added that it was probably the most uncomfortable dress ever made.

Mr. Awbery explained that besides the heat which leaked into refrigerators, products which are stored alive, such as fruit, generate heat themselves. Here preliminary experiment was necessary to guide the engineer, since different fruits breathe in, therefore producing heat at very different rates. Peaches produced a good deal of heat; then come plums; then, a long way below, apples, and finally, pears. Was it a mere coincidence that peach was used as a form of endearment and plum as one of commendation, while no such application to human beings has been made with either the apple or the pear?

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LEGAL NOTES

Horse Worked with Broken Ribs.—The allegation that a brown gelding, aged twelve years, while suffering from two broken ribs and other complaints, was beaten to make it work in harvesting on a Hampshire farm, was made at Eastleigh, on August 27th.

Harold Morris Wells, farmer, of Manor Farm, Bishopstoke, was fined £10 for permitting cruelty to the horse on August 12th, while his employee, George Moody, who had worked on the farm for 55 years, was fined £2 for cruelty to the horse, by unlawfully working it while in an unfit condition.

Mrs. Mary Taylor, of Fair Oak, said that she saw the horse, which appeared very sick and lame, beaten and dragged about 18 yards across a field to a wagon which was loaded with corn. "Moody and two other men had to drag and beat the animal to get it to move the cart along," she added.

Mr. William Maynard, veterinary surgeon, stated that a *post-mortem* showed that two ribs were badly broken. The point of one fracture was jabbing into the lung tissue, and had set up pleurisy, which had been in existence for a week. A horseman could have seen for four or five days that the animal was ill.

Moody, who pleaded guilty, told the court that he was not forced to work the horse, which went up the road all right and showed no symptoms of pain.

Mr. Wells, who pleaded not guilty, explained that this particular horse, which weighed nearly a ton, had only been in his possession for a month. It appeared to be in good condition, and it was agreed that if it was not in good condition it should be taken back to the stables. He contended that he had exercised every possible care and did all he could to save the animal pain.

. . . . .

Cruelty to a Ram.—John Winder, cattle dealer, of Hellifield, and Anthony Hunter Beck, farmer, of Kendal, were fined a total of £20 at Kendal Police Court for allowing a blackfaced ram of which they were joint owners to be cruelly ill-treated by omitting to give it proper care and attention when it was suffering in a Kendal field.

Evidence was given by an inspector of the R.S.P.C.A., who said he saw the ram banging its head on the ground. An examination revealed that a long, curling horn had grown sideways against the eyeball, which had completely gone. The empty cavity was full of maggots. The defendant Beck cut off the horn and treated the wound when the matter was pointed out to him.

A Kendal veterinary surgeon said that the ram must have been in agony for at least three or four weeks and probably for months.

Winder did not appear, but Beck, in evidence, said that the man on whose land the sheep grazed was responsible for inspecting them. The damaged eye was hidden by the curling horn.

. . . . .

Contravention of Swine Fever Regulations.—Three defendants at Salisbury Police Court were fined a total of £27, with £23 5s. in costs, for contravening swine fever regulations and the Movements of Animals Order.

Farmers gave evidence stating that as a result of defendants' pigs being sold in Salisbury market their pig herds had become infected with swine fever and between 150 and 200 animals died.

. . . . .

"Infirm" Fowls at Market.—The first prosecution under the Poultry (Exposure for Sale) Act, 1937, which came into force on July 1st, was heard at Chichester when Harry Johnson, of Maudlin, a farm hand, was summoned for exposing infirm fowls for sale in Chichester market. Richard Mason, of Shopwyke, manager of a poultry farm, was summoned for aiding and abetting.

P.-s. Catt said he noticed that five out of the nine birds were unable to stand, and an order for treatment or destruction was made.

Mr. Ernest Garry, veterinary surgeon, gave evidence as to the condition of the birds, which were stated to be suffering from fowl paralysis.

The magistrates dismissed the case against Johnson on payment of 4s. costs. Mason was fined £2 and £2 2s. costs.

* * * * *

TOXIC EFFECTS OF VASOPRESSIN IN ANIMALS

It is recalled in a recent *Lancet* annotation that attempts to relate the eclamptic toxaemias of pregnancy to over-activity of the posterior lobe of the pituitary have so far met with very little success. "One of the most obvious ways of testing the hypothesis," it is stated, "is to attempt to reproduce the symptoms and lesions of eclampsia in animals by injecting vasopressin. Some years ago Fauvet[1] claimed to have done so, but Ohligmacher[2] was unable to confirm his findings. In an attempt to decide the question Dr. Byrom[3] has been re-examining the effect of vasopressin in rats. He reports that if large enough doses of the hormone are injected gross lesions appear in various tissues. The most striking changes are found in the kidneys. About half an hour after an injection of 40 units of vasopressin these organs display large irregular areas of pallor, which later acquire the characters of anaemic arterial infarcts and are clearly due to spasm of isolated branches of the renal arteries. When smaller doses are given repeatedly for several days, a less intense and more diffuse kind of lesion is found, presumably of similar origin, which is characterised by albuminous, dropsical, or fatty degeneration of the convoluted tubules, with little or no necrosis—an appearance closely resembling the changes found in eclampsia. Eclampsia is also suggested by the occasional appearance in the liver of focal necroses, which, however, lack certain features seen in eclampsia, and by the fact that in pregnant rats vasopressin causes death and maceration of the foetuses. The parallel seems to lend some colour to the view that eclamptic lesions are expressions of vascular spasm. The dose needed to cause such lesions in animals, however, is so extremely large that Byrom finds it hard to believe that the spasm of eclampsia can be due to simple over-secretion of vasopressin, and he suggests that, if spasm is indeed concerned, it is perhaps, at least partly, due to some unknown factor that renders the vessels hypersensitive to pressor agents.

"Apart from those mentioned above, Byrom describes other lesions that can be provoked by vasopressin. Large doses repeated at short intervals are followed by gross arterial lesions, especially in the kidney, which bear close comparison with the 'necrotising arteritis' found in several human diseases, notably malignant hypertension. Focal glomerular necroses and intense hyaline-droplet degeneration of the renal tubules, also suggestive of malignant hypertension, are also occasionally found, and exceedingly large doses may give rise to the appearance of massive cortical necrosis of the kidney. It is interesting and possibly significant that both cortical necrosis and malignant hypertension are, like eclampsia, related aetiologically to pregnancy."

[1] Fauvet, E. (1931.) *Klin. Wschr.* **10**. 2125.
[2] Ohligmacher, H. (1933.) *Ibid.* **12**. 1404.
[3] Byrom, F. B. (1937.) *J. Path. Bact.* **45**. 1.

The National Pig Breeders' Association is to institute a carcase quality report scheme, complementary to the live pig shows and to the Association's carcase competitions. The scheme will be worked by arrangement with bacon factories throughout the country, and with the assistance of the Bacon Development Board. Two pigs from any litter may be sent to a factory, where measurements of the carcase will be made and marks will be allotted, and a report will be submitted to the breeder.

Correspondence

Letters to the Editor should reach the Office not later than by the first post on Tuesday morning for insertion in following Saturday's issue.

All correspondence must bear the name and address of the contributor for publication.

The views expressed in letters addressed to the Editor represent the personal view of the writer only and must not be taken as expressing the opinion or having received the approval of the N.V.M.A.

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AETIOLOGY OF CANINE HYSTERIA

To the Editor of the Veterinary Record

Sir,—May I crave the hospitality of your correspondence columns to ask the advocates of the dietetic and non-contagious theories of the aetiology of hysteria how they account for its almost world-wide distribution of a few years ago and how they would explain the following occurrence:—

In the spring of 1932 a pack of foxhounds and a pack of beagles in both of whose kennels the disease was prevalent hunted during the same week over the land of one of my clients who owns a big kennel of gun-dogs. Within a week hysteria broke out in his kennels and was prevalent during the whole of the early summer. The diet was changed several times and various methods of treatment adopted and the disease apparently died out. In August, this gentleman, who annually rents a grouse moor, took all his dogs to Scotland.

Within a week of his arrival every dog in the valley within three miles of his house—keepers', gillies' and shepherds' dogs—had contracted hysteria although none of his own then showed symptoms of it.

The district is an extremely isolated spot and all dogs in it were owned and known. These local dogs had been fed upon the same diet all their lives and the disease was previously unknown in the district.

Faithfully yours,
David Eric Wilkinson.

Oakwood,
Chigwell,
Essex.

September 11th, 1937.

Royal (Dick) Veterinary College Extension Fund.—We are informed that in the list of subscribers to this Fund published in our last issue, the subscription from Mr. W. J. Ironside, M.R.C.V.S. (£50) should have read as subscriptions in equal amounts from the following: Messrs. E. Ackroyd, W. G. Beaton, H. Burrow, K. A. Forker, E. F. Hardwick, W. J. Ironside, R. F. Montgomerie, W. J. B. Robson, C. V. Watkins, L. A. Auchterlonie, N. Bisset, N. Dobson, J. C. Grant, W. Hepburn, P. L. Le Roux, R. B. H. Murray, G. A. Sangster, J. K. Bateman, J. L. Brain, P. D. Dunn, H. W. Griffiths, G. Gowie, P. T. Lindsay, J. R. Rider and G. Tullis.

Diseases of Animals Acts
SUMMARY OF RETURNS

Period.	Anthrax.		Foot-and-Mouth Disease.		Parasitic§ Mange.		Sheep Scab.	Swine Fever.	
	Outbreaks confirmed.	Animals attacked.	Outbreaks confirmed.	Animals slaughtered as diseased or exposed to infection.	Outbreaks reported by the Local Authorities.	Animals attacked.	Outbreaks confirmed.	Outbreaks confirmed.	Swine slaughtered.
	No.	No.	No.	No.	No.	No.	No.	No.	No.
Period 16th to 31st Aug., 1937	18	23	—	—	3	4	7	27	22
Corresponding period in									
1936	8	11	—	—	4	5	9	53	31
1935	10	15	—	—	3	3	13	60	40
1934	17	28	3	361	4	4	31	41	28
Total 1st January to 31st August, 1937	490	596	19	4,401	91	129	139	748	447
Corresponding period in									
1936	312	378	4	379	86	228	163	1,356	958
1935	243	285	34	10,208	84	169	227	1,314	778
1934	264	301	5	545	98	160	409	1,079	675

Note.—The figures for the current year are approximate only.

§ Excluding outbreaks in Army Horses.